Manitoba Medical Review

Vol. 41 No. 2

February, 1961

☆

General Practitioners
VALENTINE DINNER AND DANCE
Royal Alexandra Hotel
SATURDAY, FEBRUARY 11th, 1961
See Page 119

Authorized as Second Class Mail, Post Office Department, Ottawa.

MANITOBA MEDICAL ASSOCIATION
FOUNDED 1908
QUAECUNQUE VERA

Official Publication of the MANITOBA MEDICAL ASSOCIATION Winnipeg Canada

The Manitoba Medical Review

Vol. 41 FEBRUARY, 1961 No. 2

Medicine

Clinical Diagnosis and Modern Concepts of Malabsorption*

Douglas G. Cameron, M.D. and D. G. Kinnear, M.D.

In recent years the malabsorption syndrome has become a subject of wide interest. Little more than a decade ago it was considered a rather rare condition of special academic interest but of little practical concern for the practicing physician. The exact incidence of this syndrome in Canada has not been determined, but it is being diagnosed with increasing frequency in all parts of the country. No longer can it be considered a rare disorder. The difficulty of precise diagnosis in the past probably accounted for the fact that it was seldom recognized. The progress of medical science has led to a better understanding of this syndrome. New diagnostic methods have been introduced, and effective methods of treatment are available. In these circumstances the physician must be familiar with the clinical features of malabsorption syndrome and understand the methods of investigation available to confirm the diagnosis.

Physiologic Considerations

Our knowledge of the normal processes whereby the residua of ingested foodstuffs join the body fluids is far from complete. This process of assimilation of food can be thought of as proceeding in two stages. During the first, the stage of digestion, large molecules are hydrolyzed by enzymes to smaller particles. In the second stage, the stage of absorption, these smaller particles traverse the intestinal mucosa. Faulty assimilation results when there is a defect in either stage.

Carbohydrates

The digestion of carbohydrates proceeds by the hydrolysis of polysaccharides to disaccharides by the enzyme amylase. Salivary amylase is of little or no importance in this reaction which depends directly on the amylase of pancreatic juice. Disaccharides are then split to monosaccharides by the enzymes of the succus entericus. Absorption of these sugars involves phosphorylation by specific hexokinases.

Proteins

The digestion of proteins depends on several proteolytic enzymes which break the peptide bonds binding the constituent amino acids in these large molecular aggregates. Gastric pepsin is not essential for this process. For example, in pernicious anemia the stomach fails to elaborate pepsin but defective protein absorption seldom results. The proteolytic enzymes of the pancreatic juice and succus entericus are responsible for protein hydrolysis, and impaired protein digestion results when these secretions are inadequate. Protein material crosses the intestinal mucosa as amino acids.

Fats

The digestion of neutral fat proceeds in the presence of adequate amounts of pancreatic lipase, bicarbonate and bile salts. These same three substances contribute as well to the formation of the stable emulsion which is essential for this process of fat digestion and the absorption of its end products — fatty acids and the linking glycerol molecules. However, complete hydrolysis is not an essential prerequisite to absorption. The products of partial hydrolysis, mono- or diglycerides, can be absorbed as well. In addition, some unsplit neutral fat aggregates of small particulate size in the emulsion can also be absorbed directly. Present evidence suggests that absorbed fats, glycerides and fatty acids travel in the intestinal lacteals to reach the general circulation via the thoracic duct. Glycerol is thought to travel via the portal circulation.

It is clear that the secretions of three important organs are directly concerned with the digestion and absorption of food. These are the liver, the pancreas and the small intestine. Salivary and gastric secretions are not essential. It follows that maldigestion and, as a consequence, malabsorption may result when there is insufficient delivery of pancreatic enzymes or bile salts to the small intestine. Malabsorption may result also when there is extensive pathologic changes in the small intestine, and when important segments of the gastrointestinal canal are by-passed or removed surgically. There remains a large group of patients with the malabsorption syndrome in whom the underlying cause is not clear. This idiopathic group includes patients with coeliac disease, idiopathic steatorrhea and sprue. Adlersberg has suggested the term primary malabsorption syndrome to include these three disorders[1]. Postmortem studies have failed to demonstrate clear-cut pathologic differences between them and until distinct pathologic or etiologic differences are defined, the differentiation remains a clinical one. Traditionally they have been differentiated on the basis of age, geography and response to treatment. The clinical hallmarks of sprue are fatty diarrhea, weight loss, glossitis and megaloblastic anemia in adults who have lived in the tropics. Identical clinical features may be encountered in adults with idiopathic steatorrhea,

*From the McGill University Medical Clinic of The Montreal General Hospital.

Presented at The Manitoba Medical Association Meeting, September 27th, 1960.

but these individuals have lived continually in temperate zones. Coeliac disease is a diagnosis reserved for children with idiopathic steatorrhea. Identical histologic changes are observed on jejunal biopsies in these three disorders. Moreover the pathologic change seems to be distinctive permitting differentiation from the secondary types of malabsorption. Such observations have, of course, strengthened the view that coeliac disease, sprue and idiopathic steatorrhea are variants of the same fundamental disorder, the nature of which is still obscure.

Clinical Picture

The clinical manifestations of malabsorption are protean. Reference has been made to the classical picture of sprue. Here a clinical diagnosis has rested traditionally on the occurrence of frequent bulky, pale, unformed, offensive stools, together with weight loss, glossitis and megaloblastic anemia. These same features may be encountered in the malabsorption syndrome due to other causes. In addition, hypocalcemic tetany, osteomalacia, iron deficiency anemia, volvulus of the large bowel and a hemorrhagic diathesis due to prothrombin deficiency are sometimes manifestations of this syndrome. When no other cause is apparent, any of these should suggest the diagnosis even though bowel symptoms have never been present.

The clinical features in any individual patient seem to be determined by limitations in the diet, deficient body stores or increased demands for specific nutrients as well as by faulty absorption. The defects in absorption are partial rather than complete. Consequently it is possible for a patient to remain free of symptoms for long periods if his diet contains a sufficient surplus of nutrients to compensate for the reduction in the efficiency of absorption.

The physician's challenge in the diagnosis of the malabsorption syndrome can be defined simply. It is twofold. He must suspect and then confirm that he is dealing with a malabsorption problem. Next he must determine whether it is of the primary sort or secondary to disease involving the liver, pancreas or small intestine. Clinical methods lead him to the suspicion that he is dealing with a case of malabsorption and that it is of the primary or secondary type, but he must resort to laboratory tests in order to establish the precise diagnosis.

Laboratory Tests

A wide variety of tests has been introduced to help resolve the clinical problem. By their very number these tests have confused rather than clarified the problem in the minds of many physicians. Consequently, a brief description of the more important tests and a discussion of their value is in order here.

Fat Balance Studies

The absorption of fat is always impaired in the malabsorption syndrome. Absorption of other nutrients is not constantly abnormal, and it follows that demonstration of steatorrhea is the only certain way to establish or exclude the diagnosis. A balance technique must be used and the balance period must be at least four days. Several reliable methods are available for the chemical analysis of the fecal fat. In normal adults on daily intakes of 50 to 200 gm. of fat, the fecal loss is always less than 10% of the intake. When the fecal fat exceeds 10%, one can make a secure diagnosis of steatorrhea[3].

Examination of the Stool

The silver grey color, the porridge-like consistency and foul smell of the stools in typical cases strongly suggests the diagnosis, but this distinctive sign is not always present[2]. Likewise, microscopic examination of the stools for evidence of increased fat is not entirely reliable. Inspection of the stool is a useful adjunct to the clinical examination of the patient, but impressions gained in this way are not conclusive. An experienced observer can recognize the presence of steatorrhea with considerable accuracy if there is a moderate or marked increase in fecal fat. He will often miss small increases in fecal fat and cannot ascertain the etiology of the steatorrhea with certainty[4].

Oral Tolerance Tests

Oral Tolerance Tests are probably the most widely used laboratory tests for the diagnosis of intestinal malabsorption. These tests are easy to perform, the results are available quickly and show a fair correlation with the presence or absence of steatorrhea. In these tests the blood level of the test substance is measured after oral administration of a test dose. Sometimes urinary and fecal excretions are measured as well. The rationale of these tests rests on the presumption that when malabsorption is present, blood levels of the test material will fail to reach the levels attained by normal subjects in the same time following an identical oral test dose. Unfortunately the test substances are affected unpredictably by a variety of factors such as the rates of gastric emptying, utilization, storage, release, and excretion as well as by the distribution and dilution of the test material in the blood, lymph, intra- and extracellular fluid.

It is not surprising that in any large series of patients with steatorrhea, only about three quarters will show a positive oral tolerance test with any one of the test substances. The most popular absorption tests are the glucose tolerance, vitamin A tolerance, d-Xylose tolerance, I^{131} triolein and I^{131} oleic acid tolerance tests. Typical results in normal individuals and patients with malabsorption are shown in Figure 1. The lack of a perfect correlation between the results of these tests and the presence or absence of steatorrhea is disappointing. As a consequence, the tests are of limited value in the demonstration of malabsorption. However, having

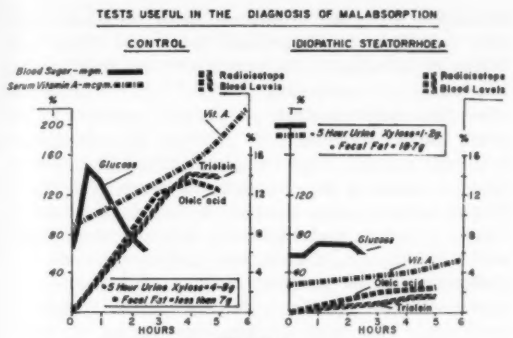


Figure 1
Oral Absorption Tests

established the presence of steatorrhea by fat balance studies, the oral tolerance tests do have a useful role in the differential diagnosis of the types of malabsorption[3].

X-ray Findings

For many years radiologists have recognized that functional alterations in the appearance of the small bowel are common in patients with malabsorption. In this connection, the most important X-ray findings in the small intestine after ingestion of barium are dilatation, segmentation and hypersecretion. Unfortunately, similar changes have been observed in a variety of conditions having no relation to malabsorption, such as hyperthyroidism, allergic states and emotional disturbances. It is true that these X-ray findings are non-specific in that by themselves they do not warrant a specific diagnosis. The indiscriminate use of terms such as "deficiency pattern" has led many physicians to mistrust, and as a consequence pay too little attention to the X-ray changes. More emphasis should be placed on the value of small intestinal mucosal detail to the physician. Most patients with malabsorption will show X-ray evidence of marked dilatation, segmentation and hypersecretion. While these findings are admittedly non-specific, they will be meaningful to the careful physician who already suspects the diagnosis and has excluded other conditions known to produce them. A few patients with primary malabsorption and malabsorption secondary to disease of the small bowel will show a normal small intestinal pattern as will patients with pancreatic steatorrhea.

A flat plate of the abdomen should precede the barium studies. Calcification in the region of the pancreas suggests a chronic pancreatic disorder and indicates the need for definitive tests of pancreatic function. Occasionally this film will reveal the demineralization of bones, pseudo-fractures or frank fractures of osteomalacia[5].

Small Bowel Biopsy

In the past few years, peroral suction biopsy of the jejunum has been established as a most important procedure in the differential diagnosis of the malabsorption syndrome. An identical histologic lesion in the proximal small bowel is present in coeliac disease, idiopathic steatorrhea and sprue. This lesion is not present when malabsorption is secondary to other disorders. Moreover, the specific histologic change in cases of primary malabsorption seems to persist when complete clinical remission is induced by modern treatment. Great care must be taken to ensure correct orientation of the fresh biopsy before it is fixed. This permits the serial sections to be cut exactly perpendicular to the mucosal surface. Tangential artefacts are obviated in this way. Normal jejunal mucosa shows delicate, discrete, tall villi covered by columnar epithelial cells whose nuclei are basal, well aligned and well organized. The lamina propria consists of a delicate framework of loose connective tissue with a light scattering of round cells. There is a striking change in primary malabsorption (Figure 2). Here, the jejunal mucosa is flattened due to shortening, blunting and clubbing of the villi. These often appear to be fused. There are fewer epithelial structures but the lamina propria is expanded and infiltrated by chronic inflammatory cells[6].

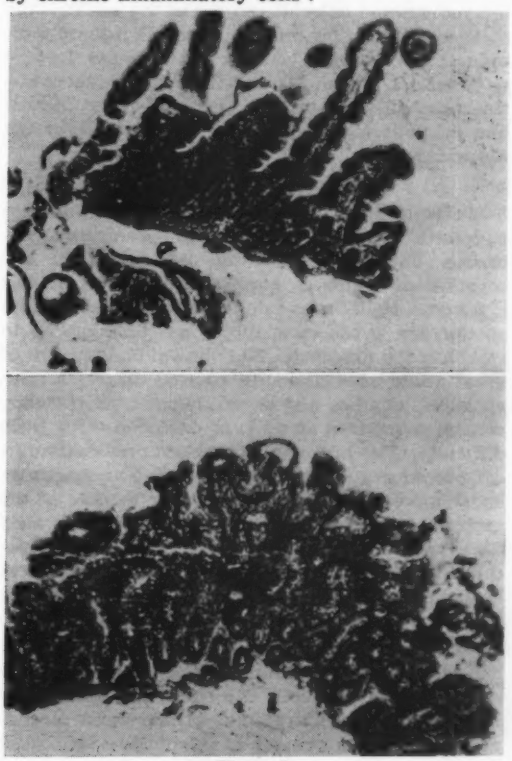


Figure 2
Jejunal biopsies
a) Normal
b) Primary Malabsorption

Secretin Test

The secretin test is the most sensitive and reliable test of pancreatic function. Under fluoroscopic guidance care must be taken to place the tip of the

double lumen tube exactly in the third or fourth part of the duodenum. A dose of secretin calculated on the basis of one unit per Kg. of body weight is injected and all secretions are collected for a period of 40 to 80 minutes. The total volume is recorded and the bicarbonate and enzyme content is determined. For practical purposes it is only necessary to estimate the amylase content for when one enzyme is diminished the others are reduced as well. Finally, the aspirates are examined for the presence of malignant cells and parasites. On the basis of different responses in volume, bicarbonate and amylase concentrations, various pancreatic defects can be differentiated. In chronic pancreatitis there is little change in the volume of secretion but the concentration of bicarbonate and amylase is reduced. A reduction in the volume of secretion with normal concentrations of bicarbonate and amylase suggests obstruction of the pancreatic ducts.

Sweat Electrolytes

It is now apparent that cystic fibrosis is not exclusively a disorder of childhood. Estimation of the sodium and chloride content of the sweat is the most specific test for this condition. In normal individuals the chloride concentration ranges from 4 to 60 mEq/L (mean 32) and the sodium concentration from 10 to 90 mEq/L (59). In cystic fibrosis one or both electrolytes are elevated above the normal range[7].

Differential Diagnosis

A careful history and physical examination may suggest a clinical diagnosis of malabsorption syndrome. Fat seems to be the only substance, absorption of which is constantly impaired in this syndrome. Hence a fat balance study to demonstrate steatorrhea is the most direct and certain way to establish the diagnosis. The clinical findings are of great value as well in differential diagnosis. Obstructive jaundice and severe hepatic insufficiency can be recognized at once or excluded with little difficulty. The history will disclose previous surgical procedures which may have led to strictures, anastomoses, extensive resection or fistulae of the gastrointestinal canal. The physician will be alert for evidence of diseases in which extensive involvement of the intestine may cause malabsorption. Lymphoma, carcinoma, Whipple's disease, amyloidosis, scleroderma and tuberculosis are well known examples.

A chest X-ray and routine blood counts may help exclude some of these. The chest film may reveal enlarged mediastinal glands, primary and secondary deposits of carcinoma, or tuberculous lesions. It may show evidence of osteomalacia. Normal blood counts tend to rule out primary malabsorption. Anemia is unusual in pancreatic insufficiency and weight loss is uncommon unless diabetes is present as well. Anemia is common in primary malabsorption and it is sometimes the presenting feature. It may be iron deficiency, megaloblastic or dimorphic in type. Examination of the bone marrow will help to determine the type of anemia. When megaloblastic anemia is encountered, a gastric analysis is indicated. Should this reveal histamine-fast achlorhydria, pernicious anemia is the most likely diagnosis. A vitamin B_{12} absorption test will usually confirm this. Should the absorption of vitamin B_{12} exceed the very low levels found in pernicious anemia, or fall in the normal range, primary malabsorption may be responsible and a fat balance is indicated. Likewise when iron deficiency is the problem and careful studies exclude occult bleeding, malabsorption must be excluded. The prothrombin time should be determined in every case. It is usually prolonged in the presence of malabsorption, sometimes dangerously so and on occasions, hemorrhage is the presenting feature. Estimation of the serum calcium, phosphorus and alkaline phosphatase levels should be a routine procedure as well. These serve to demonstrate any biochemical evidence of osteomalacia.

A careful X-ray examination of the gastrointestinal tract is often of great value. Calcification of the pancreas or unsuspected osteomalacia may be discovered in the preliminary flat film of the abdomen. The barium studies will usually show dilatation, segmentation and hypersecretion of the barium when malabsorption is present. Intestinal diverticula, stenoses, internal fistulae or regional enteritis as well as other disorders known to involve the small intestine may be identified.

At this stage of the investigation, oral tolerance tests may be useful. Pancreatic disease is suspected when the glucose tolerance curve follows the diabetic pattern, when the d-Xylose tolerance test is normal and when the I^{131} labelled oleic acid tolerance test is normal, but the I^{131} labelled triolein tolerance test shows the flat curve seen in primary malabsorption. In these circumstances a secretin test is indicated.

Biopsy of the small intestine is the most important recent development in the diagnosis of primary malabsorption. A specific lesion is found in coeliac disease, idiopathic steatorrhea and sprue. This lesion is not present in the secondary types of steatorrhea. Occasionally the biopsy will disclose the characteristic lesions of amyloidosis, tuberculosis or one of the other disease processes which sometimes involve the small intestine and lead to malabsorption.

Treatment

A few words about the treatment of malabsorption are in order, for the results have had important implications on present concepts of this syndrome. Here, as elsewhere in Medicine, precise diagnosis is an important first step towards intelligent treatment. When the malabsorption syndrome is secondary to disorders of the liver, pancreas or small intestine, the ideal treatment is specific surgical or medical therapy for the offending disease.

Unfortunately specific cures are not available for many of them. In these circumstances the physician must content himself with symptomatic therapy.

The standard treatment for primary malabsorption is largely empiric. Fundamentally, all these patients are suffering from malnutrition, but the clinical picture in individual cases is usually determined by severe deficiency of one or more specific nutrients. Traditional dietary treatment consisted of a low fat, moderate carbohydrate, high protein diet supplemented by vitamin B_{12}, folic acid, iron, vitamin K, vitamin D and calcium. This regimen produced striking clinical remissions in many, but not all cases, and relapses were frequent. Continuous fat balance studies showed that steatorrhea persisted during clinical remissions.

More recently diets free of wheat and rye gluten and sometimes barley gluten as well have been employed. In nearly all children with coeliac disease, removal of gluten from the diet results in rapid clinical improvement and continuous fat balance studies show that fat absorption returns to normal. When gluten is exhibited again, clinical relapse follows and steatorrhea returns. In adults with idiopathic steatorrhea and sprue, the same dramatic improvement sometimes follows the use of a gluten-free diet. In others, improvement may not be apparent for several months. There are also some frank failures. The results of the gluten-free diet in secondary types of malabsorption have not been fully assessed as yet, but preliminary reports suggest that it is not effective. Finally, there is some evidence that patients with primary malabsorption resistant to dietary treatment may be benefitted by the use of ACTH or one of the adrenal steroid hormones.

Discussion

The modern concept of primary malabsorption must take into account many old and several new important observations. The similarity of the clinical picture in coeliac disease, idiopathic steatorrhea and sprue has been apparent for many years. However the striking differences in age incidence, geographic distribution and response to treatment seemed to justify the view that they were different diseases. By definition coeliac disease is a disorder of childhood and only a few adult patients with idiopathic steatorrhea give a conclusive history of having suffered from coeliac disease in childhood. Moreover the dramatic response of many patients with tropical sprue to folic acid and occasionally to antibiotics, seems to differentiate it from the other two conditions. Several newer observations make it necessary to modify this older view. A high familial incidence of coeliac disease is well documented[8] and the familial incidence of idiopathic steatorrhea is not rare[9]. Moreover some adults with idiopathic steatorrhea are known to have had coeliac disease as children. It is well known that tropical sprue often affects several members of one family[10]. Previous mention has been made to the identical histologic lesion seen in jejunal biopsy in these three conditions. The lesion appears to be specific and persists during remissions. This histologic data together with the familial incidence of the three disorders has led to the suspicion that they represent different clinical manifestations of a single fundamental inherited defect.

The striking success of the gluten-free diet in coeliac disease and the prompt relapse which follows the restitution of offending glutens to the diet has led to the suggestion that susceptible individuals are allergic to these protein substances. However, there is little other evidence to support this. Certainly allergic manifestations such as eczema, hay fever, urticaria and asthma are rare in these patients. At the present time it must be concluded that the mechanism of action of gluten is unknown. Moreover the overall unpredictable results of the gluten-free diet in cases of primary malabsorption raises the possibility that the specific jejunal lesions may represent the end results of several different causes. A critical analysis of present knowledge leads to the conclusion that the dissimilarities between coeliac disease, idiopathic steatorrhea and sprue are too superficial to warrant their continued dogmatic definition as independent clinical entities. The final chapter of this mystery has yet to be written. Meanwhile the concept of primary malabsorption as a syndrome embracing these three clinical variants and due to an unexplained cause or causes is likely to serve the physician best.

References

1. Adlersberg, D.: The Malabsorption Syndrome. New York, Grune & Stratton, Inc., 1957.
2. Cameron, D. G., Bensley, E. H. and Wood, P.: Latent Steatorrhea. Ann. Int. Med., 37: 553, 1952.
3. Hunter, F. M. and Prevatt, A. L.: Diagnostic Methods in Intestinal Malabsorption. Am. J. Med. Sci., 236: 81, 1958.
4. Black, D. A. K., Bound, J. P. and Fourman, L. P. R.: Fat Absorption in Tropical Sprue. Quart. J. Med., 16: 99, 1947.
5. Marshak, R. H. and Eliasoph, J.: The Roentgen Findings in the Malabsorption Syndrome. Med. Clin. N. America, 41: 1565, 1957.
6. Rubin, C. E., Brandborg, L. L., Phelps, P. C. and Taylor, H. C., Jr.: Studies of Coelic Disease. 1. The Apparent Identical and Specific Nature of the Duodenal and Proximal Jejunal Lesion in Coeliac Disease and Idiopathic Sprue. Gastroenterology, 38: 28, 1960.
7. di Sant'Agnese, P. A., Darling, R. C., Perera, G. A. and Shea, E.: Sweat Electrolyte Disturbances Associated with Pancreatic Disease. Am. J. Med., 15: 777, 1953.
8. Thompson, M. W.: Heredity, Maternal age, Birth order in Etiology of Coeliac Disease. Am. J. Human Genetics, 3: 159, 1951.
9. Adlersberg, D.: Problems of Management of Idiopathic Sprue. New York State J. Med., 55: 3575, 1955.
10. Bahr, P. H.: Report on Researches in Sprue in Ceylon (1912-1914). London, Cambridge University Press, 1915.

Some Problems of the Acute Phase of Myocardial Infarction

T. Edward Cuddy, M.D., M.Sc.(Med.), F.R.C.P.(C)

Myocardial infarction is the admitting diagnosis in over one-third of those patients admitted to the hospital with heart disease[1]. Of the deaths in patients suffering from cardiac disease approximately 80 per cent are due to myocardial infarction[1]. With these statistics in mind it is not amiss to review the problems of the acute phase of myocardial infarction.

Approximately 35 per cent of patients admitted to hospital with a myocardial infarction die within the first six weeks[2]. The presence of one or more of the following complications are a contributory or direct cause of death in nearly all these cases. Arrhythmias, shock, congestive heart failure, myocardial rupture, and thromboembolism are some of the grave problems which complicate acute myocardial infarction and contribute to its high mortality rate. Aspects of certain of these complications with illustrative cases will be considered.

Neurological Manifestations

The diagnosis of the acute infarct is usually not a problem. Nevertheless, approximately 10 to 13 per cent of people with fatal infarcts have no symptoms of pain during an infarction and an additional 10 per cent have symptoms which are atypical[3]. The latter group may have atypical pain or may present neurological symptoms such as unconsciousness, seizural activity or hemiplegia as the admitting picture.

Case 1

The following is an example of such a situation. This 73 year old diabetic suddenly collapsed while attending a business meeting and was immediately rushed to hospital by some medical associates. On examination he was unconscious, in profound shock, with sweating, and a thready pulse. There was intermittent seizural activity, and his blood pressure was unobtainable. Hypoglycemia or massive blood loss was initially suspected, but failure to respond to 25 per cent glucose in water and a preliminary blood sugar of over 300 indicated hyperglycemia, while a normal hemoglobin and hematocrit, negative stools for blood, made the second possibility uncertain. The electrocardiograph showed a sinus rate of 88 per minute, with ischemic changes most marked in the infero-septal and posterior regions. These were not diagnostic of infarction however, and could have followed the prolonged period of hypotension. The patient regained consciousness following the administration of oxygen, respiratory and cardiac stimulants. On regaining consciousness, the patient admitted to some abdominal discomfort. A stable blood pressure, pulse, and hematocrit followed and further belied concealed blood loss.

Serum glutamic oxalic transaminase during the next four days showed a course characteristic of myocardial infarction. It was 550 units sixteen hours after the acute episode and fell to 320 the second day, and 85 the third day. Consonant with the enzyme elevation, serial electrocardiographs showed progressive loss of R's in the infero-septal region providing electrical confirmation of the presence of myocardial necrosis. This man subsequently made an uneventful recovery.

This case serves to illustrate that acute vascular collapse may result from myocardial infarction and may precipitate unconsciousness and epileptiform seizures. The collapse fortunately may be reversible even though it is not transient. On the other hand, one should suspect the possibility that a patient presenting with an acute hemiplegia may have suffered cerebral embolization, secondary to dislodging of a mural thrombus from the site of a recent infarction. It also serves to demonstrate the usefulness of serial electrocardiograms and serum enzyme levels in those cases presenting diagnostic difficulties. This case had a serum glutamic oxalic transaminase level which was 12 times the normal, and the return to normal in four to six days is characteristic of those changes found in an acute myocardial infarction of a fairly large size. Similarly, the lactic acid dehydrogenase or L.A.D. is found elevated, while the serum glutamic pyruvate transaminase is nearer normal[4]. This is in contra-distinction to what is found in liver disease where the latter (S.G.P.T.) is higher than the former two, all three remaining elevated during the course of the hepatic disease.

Shock

Shock, clinically apparent by collapse, with hypotension, absent venous filling, sweating or ashen cyanosis occurs in approximately 10 to 20 per cent of the diagnosed cases of myocardial infarction[5]. When it persists longer than one hour, and is unrelieved by ordinary supportive therapy such as control of pain and oxygen, it carries a mortality rate of 80 to 100 per cent. Vasopressor therapy, notably norepinephrine has been given as an intravenous infusion. Under such circumstances 4 cc, or one ampule, are diluted in 1000 cc of glucose in water, or a stronger solution is used if the administration rate of 50 drops per minute fails to raise the blood pressure. The results of this treatment vary. Thus, of 131 patients treated in this way, the mortality has been variously reported from 14 to 100 per cent[6]. The average mortality — 60 per cent, suggests some improvement over the untreated group, although the two groups are often dissimilar in severity, duration, or other characteristics. In our own experience there has been 100 per cent mortality in the patients treated with norepinephrine. The only survivors to date belong to the untreated group.

From the Department of Medicine of the University of Manitoba and the Clinical Investigation Unit of the Winnipeg General Hospital.

Case 2

We had the opportunity to see and study a 58 year old man who presented with a classical history of infarction which the electrocardiograph localized to the infero-septal region. His blood pressure was not obtainable for over six hours, although a faint femoral pulse could be ascertained. Because there were basal rales and distended neck veins he was given digitalis. Cardiac outputs were determined using the dye dilution technique and the femoral artery was punctured and used for sampling. His cardiac index was very low, measuring 0.73 1/min/M². (The normal is 3 to 4 1/min/M²). The femoral artery pressure was 110/65 at the time that pulses were absent and cuff pressures were not obtainable. His peripheral vascular resistance, as you might expect, was extremely high. This man survived without vasopressor therapy, although intravenous glucose and water was given at a rate similar to levophed administration. His hemodynamic findings were similar to others reported in this condition[7] and suggest that marked vascular constriction is already in progress without the introduction of exogenous vasopressors. A similar study employing vasodilator therapy (adrenergic blockade) is planned for the future.

Cardiac Arrythmias

Cardiac arrythmias are a common occurrence in acute myocardial infarction developing in roughly 20 per cent[8]. These cannot be considered benign, for even a sinus tachycardia persisting for 24 hours or more carries a mortality double that in patients without this complication. In addition 82 per cent of patients with frequent extrasystoles die[9], giving mute evidence that extrasystoles are often forerunners of fatal ventricular arrhythmias. Atrial fibrillation is a common arrhythmia, and is found in 7 to 9 per cent of cases. Even when transient, the mortality rate is increased, and when persistent, it is associated with a mortality of 89 per cent[10]. Paroxysmal atrial tachycardia is less frequent, though equally serious, as is borne out by the following case.

Case 3

A 47 year old man had been seen with an acute infarction two years previously. During the acute stage, he developed paroxysmal atrial tachycardia with a rate of 160. **Figure 1A**, is an electrocardiogram taken in 1956, and shows a rapid but regular atrial and ventricular rate. The QRS is broad due to a right bundle branch block. The supraventricular origin of the rhythm is evidenced by the P waves which precede each QRS in leads III, V_1 and V_2. The changes indicative of anterior infarction are also present. This arrythmia was successfully treated with digitalis and oral quinidine, and the patient recovered. Despite continuous anticoagulant therapy, he was re-admitted to hospital during August of 1960 with a recurrent anterior myocardial infarction. The next morning he was found in shock with a blood pressure of 90 to 100 systolic and a completely regular apical rate of over 180. He had a very loud gallop rhythm. **Figure 1B** which shows the electrocardiogram taken in August, 1960, illustrates a remarkable similarity to the previous arrythmia. The broad abnormal-looking QRS is again due to a right bundle branch block and P waves can be seen in the first two precordial leads. Levophed (norepinephrine) was administered intravenously to maintain his blood pressure. Despite full dosage of digitalis, and both intramuscular and oral quinidine, up to a maximum of 50 to 60 grains per day, this rhythm could not be interrupted. **Figure 1C** which illustrates the record taken six days later, shows slowing of the rate to 160 and definitely confirms the supraventricular origin of the arrhythmia, P waves being seen in leads III, AVF, V_2 and V_3. This man died eight days later without antecedent change in his rhythm.

Ventricular arrhythmias are generally fatal in 85 per cent or more of cases of myocardial infarction, in which they occur.

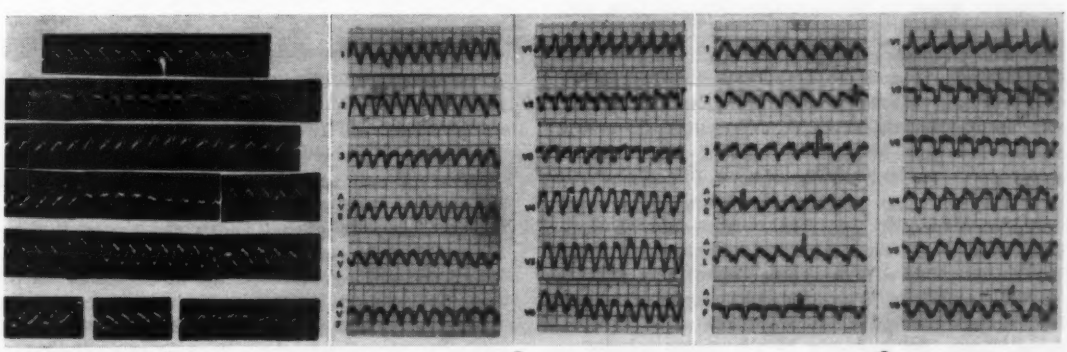


Figure 1 — The Electrocardiograph of Case 3

A. Electrocardiograph of December 28, 1956, shows a complete right bundle branch block, anterior myocardial infarction and supraventricular tachycardia — rate 160. B. Record of August 22, 1960, illustrating supraventricular tachycardia — rate 180, RBBB and anterior-lateral myocardial infarction. C. Record of August 29, 1960, similar to B — slowing of rate to 160.

Ventricular Asystole

Sudden death is frequent in acute myocardial infarction, occurring in about 25 per cent of patients before they reach hospital and in an additional 10 per cent of those surviving admission to hospital[1]. Although some of these fatalities are due to ventricular fibrillation, it is known that many of these deaths result from complete A-V block with ventricular asystole, which in turn may lead to ventricular fibrillation. The more diligent the supervision of patients with recent infarcts, the more numerous are the cases with recognized asystole, and more important, the more successful is the therapy of this complication. Electrical monitoring equipment may help in the special ward or clinic, although there are often no certain clinical indications preceding the potentially fatal arrhythmia to indicate which patients require monitoring. Automatic electrical pacing of the heart can be performed externally, following any preselected interval of asystole when such equipment is used. It is conceivable that 10 per cent of people dying of acute myocardial infarction could be saved if all cases were so monitored. Short of this, however, all cases with any evidence of conduction defects, with frequent extrasystoles, or with extremes of pulse rate, should be closely followed, and electrically monitored, if such equipment is available. If ventricular fibrillation is detected early enough, external defibrillation can be carried out successfully. However, if the duration of fibrillation is longer than a minute or two, cardiac massage is a necessary prerequisite to defibrillation. A new method of external cardiac massage, suggested first from Oklahoma[11], has been fully described recently from Baltimore[12]. This method is illustrated graphically in Figure 2. This illustrates the position of the patient, lying supine on a firm surface with the attendant exerting firm pressure with both hands over the lower end of the sternum in a rhythmic fashion at a rate of approximately 60 per minute. This is reported to be effective in pumping adequate blood in both unconscious children and adults in whom asystole or fibrillation has developed. In such patients, sufficient flexibility of the rib cage is said to permit the compression of the heart against the vertebral column posteriorly. We have used this method effectively in a few cases.

Figure 2

An illustration of external cardiac massage in an adult. Note the position of the patient, supine on a firm support. An airway and assisted ventilation are necessary. Note also the position of the administering hands with the flat of the palm over the lower end of the sternum and additional pressure from the superimposed hand.

Case 4

A final case may illustrate the effective management of a case of recurrent asystole due to complete A-V block. This 78 year old man was admitted April 19, 1960, with a typical history of a myocardial infarction (Fig. 3A). The next day he still had minor chest pain, but was reasonably comfortable. Later, he was seen by the senior interne to have become suddenly pale, sweaty, and convulsing. During this time he was pulseless, but his pulse soon returned to a rate of 25 to 30 per minute. Resuscitation could be accomplished by pounding over the sternum. An electrocardiograph revealed a complete A-V block with a very slow ventricular rhythm (Fig. 3B). This record shows complete A-V dissociation, an atrial rate of 115, and a ventricular rate of 25 per minute.

Two immediate measures were undertaken. The pacemaker-monitor was immediately set up. Three electrodes were placed across the precordium so that if asystole persisted for longer than six seconds, electrical pacing of the heart at a rate of 80 per minute ensued automatically. This equipment safe-guarded the patient's life while an intravenous infusion of isoproterenol (isuprel) was established, (0.4 mg were diluted in one liter of glucose in water). Figure 3C shows that the infusion rate was gradually increased in the first and second strips until a ventricular response of approximately

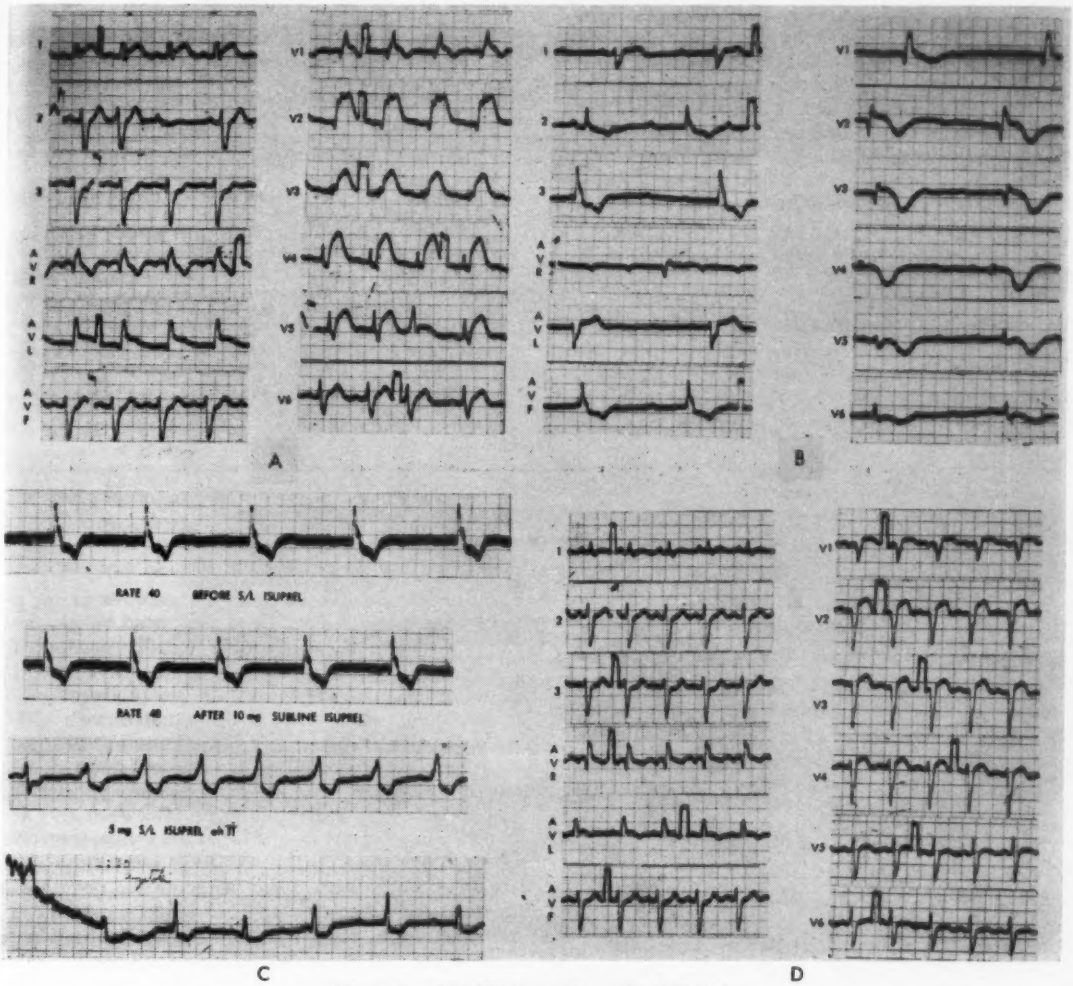


Figure 3 — The Electrocardiograph of Case 4

A. April 19, 1960 — electrocardiograph taken on admission. Anterior myocardial infarction and right bundle branch block. Occasional ventricular extrasystole. B. April 20, 1960. Complete A-V block. Atrial rate 95. Ventricular rate 42. C. Successive EKG records April 21, 1960, during the administration of isuprel as indicated. Complete A-V block persists. D. Normal A-V conduction.

50 per minute was maintained. Later, 10 mg of isuprel were given sublingually every hour. This patient was quite susceptible to the drug and the ventricular rate became rapid, going up to 70 to 90 per minute, and occasional ventricular extrasystoles were seen, such as in strip three. The dosage was then reduced, but the patient had another period of asystole, which was treated by the pacemaker. This episode is shown on the bottom strip of Figure 3C. Finally a maintenance dosage level of 5 mg every two hours was established, and a satisfactory ventricular rate was maintained. The very next day, A-V conducting returned to normal (Fig. 3D). Despite a sinus tachycardia, this man survived and is currently doing well on continuous anticoagulants.

Adrenal cortical steroids such as cortisone or hydrocortisone have been used successfully in reversing complete A-V block in recent myocardial infarction[13]. Similarly, the use of molar sodium lactate has resulted in effective ventricular rates in ventricular asystole[14].

In the future, it is conceivable that either asystole or ventricular fibrillation can be successfully diagnosed and treated by external methods without recourse to thoracotomy and its attended complications. It should be emphasized that cardiac resuscitation demands an adequate airway and assisted ventilation, by mouth to mouth breathing or mechanical devices, to ensure adequate oxygenation. It is a fact that it is impossible to resuscitate a hypoxic heart.

There are other dreaded complications of acute myocardial infarction such as myocardial or septal rupture but space does not permit their presentation. I hope the foregoing discussion has indicated the importance of recognition and management of some of the complications of acute myocardial infarction which are amenable to treatment.

Acknowledgments

I am indebted to Drs. R. E. Beamish, B. B. Fast and J. D. McDowell for permission to publish their cases. Thanks also are due to Dr. Kouwenhoven and the Journal of the American Medical Association for permission to reprint Figure 2.

References

1. Wood, P.: Disease of the heart and circulation. Eyre and Spottiswoode, 1956.
2. Honey, G. E. and Truelove, S. C.: Prognostic factors in myocardial infarction. Lancet, 1: 1155, 1957.
3. Johnson, W. J., Achor, R. W. P., Burchell, H. B. and Edwards, J. E.: Unrecognized myocardial infarction. A clinicopathologic study. A.M.A. Arch. of Int. Med., 103: 253, 1959.
4. Wroblewski, F.: The clinical significance of serum enzyme alterations associated with myocardial infarction. Am. Heart J., 54: 219, 1957.
5. Griffith, G. C., Wallace, W. B., Cochran, B., Jr., Nerlick, W. E. and Frasher, W. G.: The treatment of shock associated with myocardial infarction. Circulation, 9: 527, 1954.
6. Malach, M. and Rosenberg, B. A.: Acute myocardial infarction in a city hospital, III. Experience with shock. Am. J. Cardiol., 5: 487, 1960.
7. Freis, E. D., Schnaper, H. W., Johnson, R. L. and Schreiner, G. E.: Hemodynamic alterations in acute myocardial infarction. I. Cardiac output, mean arterial pressure, total peripheral resistance, "central" and total blood volumes, venous pressure and average circulation time. J. Clin. Invest., 31: 131, 1952.
8. Pardue, W. O.: Arrythmias in acute myocardial infarction — a brief review. Am. Pract. Digest. Treat., 10: 810, 1959.
9. Woods, R. M. and Barnes, A. R.: Factors influencing immediate mortality after acute coronary occlusion. Am. Heart J., 24: 4, 1942.
10. Askey, J. M. and Neurath, O.: The prognostic significance of atrial fibrillation in association with myocardial infarction. Am. Heart J., 29: 575, 1945.
11. Stout, A. J.: Cardiac arrest: massage without incision. J. Oklahoma M. Assoc., 3: 112, 1957.
12. Kouwenhoven, W. B., Ing, Jude, J. R. and Knickerbocker, G. C.: Closed-chest cardiac massage. J.A.M.A., 173: 1064, 1960.
13. Phelps, M. D. and Lindsay, J. D., Jr.: Cortisone in Stokes-Adams disease secondary to myocardial infarction. A report of a case. New Engl. J. Med., 256: 204, 1957.
14. Bellet, S., Wasserman, F. and Brody, J. E.: Treatment of cardiac arrest in slow ventricular rates and incomplete A-V heart block. Use of molar and half molar sodium lactate: clinical studies. Circ., 11: 685, 1955.

Pulseless Disease
A Review

W. B. Chodirker

Pulseless Disease is a specific disease entity occurring mainly in young females and characterized clinically by absent pulses and ischaemic manifestations in the upper limbs, head and neck. The pathology consists of a specific primary arteritis with occlusion of the aortic arch and the vessels arising from it. Takayasu, a Japanese ophthalmologist drew attention to this syndrome by describing the ocular manifestations in 1909. Since then cases have been reported from many countries, but. because of inadequate documentation and confusion with other aortic arch syndromes, the number of authentic case reports in the literature cannot be tabulated[1, 3, 5, 6, 7, 16, 17, 20, 21]. Confusion has also arisen in nomenclature as several names (Aortic Arch Syndrome, Takayasu's Syndrome, Young Girl Arteritis, Reversed Coarctation, Branchial Arteritis, etc.) have been used in reference to many lesions in the rgeion of the aortic arch. The term aortic arch syndrome should be applied to any condition characterized by absent pulses and ischemic manifestations in the upper extremity and head due to pathology involving the aortic arch and its branches. The entity described by Takayasu is one of the aortic arch syndromes and for historical reasons is best referred to as Pulseless Disease until such time as a more specific name is suggested.

This paper is a summary of knowledge relating to Pulseless Disease with particular reference to recent literature.

Clinical Picture

The disease is most common in females 9-45 years of age who present with a history of transient neurological and vascular abnormalities of an ischemic nature referable to the head, neck, and upper limbs. These episodes usually occur with increasing frequency and severity over several years until permanent deficits are produced. The protracted course may be punctuated with spontaneous remissions and exacerbations, but the overall course is downhill with ultimate death due to neurological and trophic defects, cerebro-vascular accidents or cardiac failure. Occasionally the condition may be fulminant with a fatal outcome within a few months of the onset of symptoms.

The clinical picture as it presents and develops is extremely variable, but all signs and symptoms are explicable on the basis of vascular obstruction of the aorta and its major branches and are referable to the central nervous system, eyes, face, extremities and cardio-vascular system directly[12, 13]. In addition systemic manifestations occur.

The cerebral abnormalities are the result of occlusions of the common carotid arteries near their origins. Because of the free anastamoses with the vertebral vascular system which is not involved in the pathological process, symptoms usually do not occur until the carotid system is almost completely occluded. Dizziness and vertigo, giddiness and syncope, headaches and convulsions are common. Hemiplegias, quadraplegias, aphasias and a multitude of other less widespread neurological abnormalities, transient and recurrent at first eventually become irreversible. Mental impairment and auditory disturbances occur occasionally.

The most characteristic visual manifestations[4, 8], are recurrent episodes of transient blurring or loss of vision lasting seconds or minutes and usually precipitated by changing from the reclining to the upright position. Scotomata and photophobia may

be associated. These attacks which occur early in the clinical course of the disease are due to an acute reduction in retinal artery blood flow and are characterized funduscopically by a sluggish retinal arterial blood flow and fragmentation of the vascular stream. With progression of the disease irreversible intraocular changes become evident, the most characteristic of which are cataracts and peripapillary arterio-venous anastamoses of the retinal arteries. Retinal hemorrhages, dilated retinal vessels and capillary micro aneurysms also occur. In advanced cases retinal and optic atrophy develops as vision is completely lost.

Facial ischemic manifestations include thin pigmented skin, atrophic facial musculature, atrophy of alveolar processes with loss of teeth, ulceration of the hard palate, perforated nasal septum and saddle nose and alopecia. Claudication of the masticatory muscles has also been reported.

The characteristic upper limb findings present in all cases of Pulseless Disease are diminished or absent pulses with low or unobtainable blood pressure. Abnormal radial, brachial or carotid pulses and differing blood pressures in the two arms has been reported as an isolated finding several years before the onset of symptoms. Despite almost complete occlusion of the subclavian arteries the anastamotic channels are so rich that few complaints and only minimal signs of vascular insufficiency are referable to the upper limbs. Trophic finger nail changes and slight muscle atrophy are occasionally found. Claudication is rare.

Ischemic changes in the lower limbs are unusual findings but do occur. A well documented case of Pulseless Disease with iliac arteritis has been reported[11]. Elevated blood pressure in the lower extremities is a frequent finding and with the absent pulses in the upper extremities has suggested the name Reversed Coarctation for the syndrome.

The cardio-vascular findings in Pulseless Disease are the result of a greatly increased collateral circulation. Grossly enlarged superficial collateral vessels are commonly present on the chest, neck and arms, and rib notching may be palpated or seen on roentgenographs. Systolic and diastolic murmurs heard at the base of the heart and in the supraclavicular areas are due to the rushing of blood through the collaterals and may simulate the murmurs of patent ductus arteriosus, aortic insufficiency and arterio venous fistulae. A hypersensitive carotid sinus due to involvement of the sinus by the arteritis may result in bradycardia and may be a factor in the production of the syncopal attacks. Angina pectoris, myocardial infarction and congestive heart failure may occur late in the course of the disease.

An elevated erythrocyte sedimentation rate is a very frequent finding. Low grade fever, leukocytosis, elevated gamma and alpha-2 globulin have also been reported.

Pathological Features

The pathological features[10, 12, 13, 14], are characteristically found in the arch of the aorta, the common carotid arteries to their bifurcation, and the innominate and subclavian arteries. Involvement of thoracic and abdominal aorta and pulmonary, coronary, intercostal, mesenteric and iliac arteries near their origin may occur, but involvement of vertebral, brachial and cerebral vessels has not been reported.

Due to a fibrotic process the involved vessels are thickened and rigid with a narrowed lumen. An organizing thrombosis, an inconstant feature, may result in complete occlusion of the aortic branches, but, although the pathology is most advanced in the aortic arch, complete occlusion is rare. Histologically the lesion consists of a periarteritis which progresses to a panarteritis with lymphocytic and plasmacytic infiltration and is followed by cicatrization. All layers of the vessel wall are involved. Foreign body giant cells have been reported in the media, but are an infrequent finding. There is no fibrinoid or hyaline degeneration and no atheromatous changes.

Differential Diagnosis

The finding of disconnected transitory but recurring optic and neurological abnormalities may suggest a diagnosis of disseminated sclerosis[15]. This is precluded however by the discovery of absent upper limb and neck pulsations which, in the absence of some obvious explanation, labels the condition an aortic arch syndrome. These are differentiated on the basis of clinical, pathological, radiological (aortography and arteriography partciularly) and laboratory findings. The most distinguishing features of the conditions to be differentiated from Pulseless Disease are as follows[13, 16].

Congenital aortic anomalies may present with one or more absent limb or neck pulses at an early age. Both, ischemic manifestations and neurological abnormalities are absent. The diagnosis is established by aortography.

Aneurysms of the ascending aorta are most common in elderly people and are usually luetic in origin. Ischaemic changes are referable almost exclusively to the heart. Diagnosis is established by the distinctive roentgenographic appearance and the positive serological test for Syphilis.

Chronic dissecting aneurysms occur in middle aged and elderly hypertensives with a history of excruciating chest and back pain preceding the finding of diminished upper limb pulsations. Widening of the aorta is a common roentgenographic feature.

Arteriosclerosis with signs of vascular insufficiency to the upper limbs and head is usually the result of a sudden aortic thrombosis. It occurs almost exclusively in elderly individuals and is usually associated with severe coronary artery disease. The major branches of the aorta are rarely involved in the thrombotic process.

Thromboangiitis obliterans is a thrombotic arteritis with thrombophlebitis of the medium sized vessels of the legs which occurs almost exclusively in males and usually results in gangrene with loss of the limb. Excruciating pain is characteristic. Histologically fibrinoid degeneration is evident. Upper extremity involvement is exceedingly rare.

Polyarteritis Nodosa is a generalized disease of the small arteries and arterioles occurring mainly in males and characterized histologically by fibrinoid degeneration. Symptomatology is widespread with renal, pulmonary, cardiac and arthritic manifestations.

Temporal arteritis is considered a form of Polyarteritis Nodosa occurring in elderly males and involving the temporal arteries. Severe throbbing cranial pain is characteristic.

Many other diseases may manifest as aortic arch syndromes. These include Rheumatic Arteritis, Ergotism, Disseminated Lupus Erythematosus, Necrotizing Angiitis, ball thrombus of left ventricle with arterial embolism, mediastinal tumors, and Pseudomyxoma Elasticum.

Etiology

The etiology of Pulseless Disease, only recently recognized as a distinct pathological process, is unknown. The clinical and pathological pictures are not compatible with any established disease process. An infectious etiology, long suspected, remains unproved, and, although many theories have been postulated, none are supported by positive evidence and none are free of serious objections.

Early writers considered the condition tuberculous[19], or luetic but the pathological picture resembles neither condition; tubercle bacilli have never been recovered from the lesions and the serological test for syphilis is negative.

The frequent association of Pulseless Disease with preceding streptococcal infections and allergic phenomena such as erythema multiforme and erythema nodosum has suggested hypersensitivity as an etiologic factor[2]. Accordingly, it has recently been considered with the collagen group of diseases. This is supported by the finding of elevated erythrocyte sedimentation rate and high gamma globulin levels and the reported favorable clinical response to corticosteroids. Against this concept however is the distinct histological appearance without the characteristic features of the collagenoses, fibrinoid degeneration.

An endocrine etiology is suggested by the predominant female incidence and by the reported (but questionably significant) high incidence of pregnancy shortly before the onset of symptoms[2]. More direct evidence in support of this view is, however, unavailable.

Thus, pending further investigation, Pulseless Disease can be classified only in the nebulous miscellaneous group of idiopathic diseases.

Treatment

At present the treatment of Pulseless Disease is unsatisfactory. Promising results have recently been reported in one case treated with corticosteroids[14], and in another treated with anticoagulants in addition to corticosteroids[11]. Considerable further experience in the use of these drugs is necessary however before any judgment of their efficacy may be made. Androgens, antibiotics and vasodilators have not altered the clinical course of the disease. Surgical approaches[9, 19], to the treatment of aortic arch syndromes include thrombectomy, sympathectomy, carotid sinus excision, arterial grafting and by-pass procedures. Although marked improvement in the clinical condition may be produced, the operative mortality is high and the underlying pathological process of Pulseless Disease is unaltered.

Summary

Pulseless Disease is a specific arteritis of the aortic arch and its major branches occurring primarily in females and usually running a protracted but ultimately fatal course. The disease is characterized by absent upper limb and neck pulses, signs of a collateral circulation and ischemic manifestations in the upper extremities, face, eyes and central nervous system. The etiology is unknown. The disease is differentiated from other aortic arch syndromes by clinical and pathological characteristics and radiographic and laboratory findings. There is no specific treatment although promising results have been obtained with corticosteroids and anticoagulants.

Bibliography

1. Aravanis, C., Avogonstakis S., Michaelides, G.: Pulseless Disease — Reports of a Case. Am. Heart J., 56:126, 1958.
2. Ask-Upmark, E.: On the Pulseless Disease Outside of Japan. Acta. Med. Scand., 149: 161, 1956.
3. Barker, N. W., Edwards, J. E.: Primary Arteritis of the Aortic Arch. Circulation, 11: 846, 1955.
4. Benedict, W. L., Wagner, H. P.: The Ocular Lesions of Pulseless Disease. Am. J. of Med. Sc., 235: 220, 1958.
5. Birke, G., Esrup, B., Olhagen, B.: Pulseless Disease — A Clinical Analysis of Ten Cases. Angiology, 8: 433, 1957.
6. Bursten, J., Lindstrom, B., Wasastjerma, C.: Aortic Arch Syndrome — A Survey of Five Cases. Acta. Med. Scand., 157: 365, 1957.
7. Caccamise, W. C., Whitman, J. F.: Pulseless Disease — A Preliminary Case Report: 44: 629, 1952.
8. Caccamise, W. G., Okuda, K: Takayasu's or Pulseless Disease — An Unusual Syndrome with Ocular Manifestations. Am. J. Ophthalmology, 37: 784, 1954.
9. DeBakey, M. E., Morris, G. C. Jr., Jordon, C. L., Cooley, D. A.: Segmental Thrombo-Obliterative Disease of Branches of Aortic Arch. J.A.M.A., 166: 998, 1958.
10. Frouig, A. G., Loken, A. C.: The Syndrome of Obliteration of the Arterial Branches of the Aortic Arch, Due to Arteritis. Acta. Psych. et Neur. Scand., 26: 313, 1951.
11. Gibbons, I. B., King, R. L.: Obliterative Branchiocephalic Arteritis. Circulation, 15: 845, 1957.
12. Kalmansohn, R. B., Kalmansohn, R. W.: Thrombotic Obliteration of the Branches of the Aortic Arch. Circulation, 15: 237, 1957.
13. Koszewski, B. J.: Branchial Arteritis or Aortic Arch Arteritis, a New Inflammatory Arterial Disease (Pulseless Disease) Critical Review. Angiology, 9: 180, 1958.
14. Koszewski, B., Hubbard, F.: Pulseless Disease Due to Branchial Arteritis. Circulation, 16: 406, 1957.
15. Lessof, Maurice: Pulseless Disease. Guy's Hosp. Reports, 107: 53, 1958.
16. Lomas, R. W., Bolande, R. P., Gibson, W. M.: Primary Arteritis of the Aorta in a Child. A.M.A.J. of Diseases of Children, 97: 87, 1959.
17. Mirsa, S. S., Prakash, S., Agrawal, P. L.: Pulseless Disease. Am. Heart J., 5: 177, 1959.
18. Ross, R. S., McKusick, U. A.: Aortic Arch Syndromes. Arch. Int. Med., 92: 701, 1953.
19. Shimizu, K., Sano, K.: Pulseless Disease (Abstract). J.A.M.A., 145: 1095, 1951.
20. Warren, R., Triedman, L. J.: Pulseless Disease and Carotid Artery Thrombosis Surgical Considerations. New Eng. J. Med., 257: 685, 1957.
21. Weir, A. B., Kyle, J. W.: Reversed Coarctation—Review of Pulseless Disease and Report of a Case. Ann. Int. Med., 45: 681, 1956.

Surgery

The Management of Complicated Malignant Lesions of the Colon

Claude J. Hunt, M.D.
Kansas City, Missouri

I wish to discuss two of the major complications that are encountered in the surgical management of cancer of the colon. They are those associated with fixation and local extension of cancer and those related to obstruction.

The complications that are associated with malignant lesions of the colon are largely related to the segment of the colon involved. Lesions located in the right colon produce symptoms and complications entirely different from those of the left colon. This is due to the difference in the anatomy and physiology of the two colons. The right colon is derived from the mid gut and has a physiology much like that of the lower small bowel. Its content is semi-fluid and it has the property of water absorption. The anatomy is different in that the caliber of the bowel is larger and the musculature is less pronounced than with the left colon.

The malignant lesions that are encountered in the various segments of the colon, therefore, have physical characteristics peculiar to their anatomic and physiologic environment. Lesions, correspondingly, on the right side grow well out into the lumen of the bowel while those on the left side form either by an infiltrating adenomatous growth, a constricted lumen, or by lymphatic infiltration in the wall of the bowel, an annular or canalizing obstruction. The symptoms and complications are, therefore, different.

Right Colon Inflammation and Obstruction

Right colon lesions are ulcerative growths that often break down, bleed and become secondarily infected. Marked anemia develops, and from the absorption of the by-products of protein decomposition and the associated infection the lemon color of cachexia is often seen. This is a characteristic of right colon lesions, which by no means indicates extension and inoperability, as it would were it associated with a left colon cancer.

Extrinsic inflammation and edema may result from this process of necrosis and secondary infection. Penetration and perforation may result in abscess formation, often difficult to distinguish from an appendiceal abscess or diverticulitis.

These associated complications of right colon cancers rarely are so pronounced that resection cannot be done as a primary procedure after proper preoperative preparation. Blood loss can be restored by adequate transfusions of whole blood and infection can be diminished by thoroughly emptying the alimentary tract from above, by cleansing enemas, by penicillin, streptomycin and intestinal inhibitory sulfa drugs. These are routine preliminary procedures in all nonobstructive colon lesions, whether on the right or left side. If fever is present, an increased white blood count, evidence of local tenderness, or a fixed mass, a more protracted and intensive period of preparation is demanded. The Miller-Abbott tube may be helpful in reducing small bowel distention, if present, and to put the bowel at rest.

The true nature of the lesion is not, however, known until the abdomen is opened and the tumor mass examined. On the right side it may occasionally be found that a preliminary side-tracking operation, in the nature of an ileocolostomy is advisable.

Appreciable obstruction of the hepatic flexure may occur from edema, retraction and angulation without extrinsic malignant extension and respond to intestinal intubation and conservative medical measures, and ultimately be primarily resectable.

Splenic Flexure

Complications are often associated with splenic flexure cancer. The lesion is usually advanced, often fixed and obstructed before the patient is seen by the surgeon.

The symptoms before obstruction are vague and abdominal in character. The lesion is high under the costal arch and often not palpable.

A barium enema is often misleading as the proximal limb of the flexure may override or be anterior to the distal limb and show no deformity in a non-obstructive tumor. This relationship results from a high fixed contracting lesion which has pulled the two limbs of the flexure in this relationship. Therefore, only by an oblique exposure can the splenic flexure be well visualized and the two superimposed segments be distinguished.

Descending Colon and Sigmoid

Lesions of this portion of the colon are usually annular or more or less constricting in character, and at the time of consultation have a variable degree of chronic obstruction or complete obstruction. Fortunately, most apparent obstructions are not complete neoplastic obstructions, but are in part due to associated edema, inflammatory reaction and invaginated mucous membrane into the narrowed neoplastic lumen. Under proper surgical decompression and by irrigations they relent sufficiently so that the colon may partially empty itself through the distal segment. Proximal surgical decompression can be done by cecostomy or transverse colon colostomy, the latter being preferred by most surgeons. In complete obstruction with extensive proximal bowel distention transverse colon colostomy is not always easy. Often the distended right transverse colon is pushed high

Presented at the Annual Meeting, International College of Surgeons, Canadian and United States Sections, Marlborough Hotel, Winnipeg, September 28th, 1960.

under the liver by the enormously distended cecum, which pushes everything well toward the midline and upward, so the transverse colon is unapproachable.

The point of greatest clinical interest is that colon obstruction is a closed loop obstruction and is subject to gangrene and perforation when the intra colonic pressure equals or exceeds the systolic blood pressure. In this way, colon obstruction is comparable to strangulated small bowel obstruction, as no colon contents regurgitates back into the terminal ileum and distention progressively increases with ultimate impairment of the blood supply. Therefore surgical decompression should not be long deferred, else gangrene and rupture of the cecum will occur. We have observed this twice, and in one instance, it developed pending preparation for operation. Cecostomy, however, was immediately carried out, and fortunately there was little abdominal soiling and recovery occurred and subsequent resection of the lesion was successfully done.

A spontaneous rupture of the sigmoid into the free abdominal cavity was observed also in a male of 21. On lifting, a severe abdominal pain occurred and he was sent to the hospital with a diagnosis of a perforated duodenal ulcer. The abdomen was board-like. Operation revealed no ulcer perforation. The abdomen was contaminated and a pendulous sigmoid easily revealed a perforation of an indurated lesion of the anti-mesenteric side of the bowel. This was exteriorized through a left pararectus incision and obstructively resected. A complementary cecostomy was done to prevent intestinal distention and to make less favorable the development of peritonitis. A Miller-Abbott tube was passed. It is noted that the peritoneum can withstand much contamination if intestinal distention is prevented. The patient recovered. The bowel opening was later closed and the patient has been well now over fifteen years.

Rectosigmoid

The anatomic classification of the rectum, rectosigmoid and extreme lower sigmoid is not very clear. The rectosigmoid can not be anatomically defined. The excised specimen shows no such anatomic structure. We think it is more appropriate to describe carcinoma of that portion below the pelvic peritoneum as "extraperitoneal carcinoma" and that above and below the promontory of the sacrum as "intraperitoneal."

Lesions below the pelvic peritoneum we often believe are unsuitable for excision and bowel reconstruction. The problem always is, can you remove all the cancer and can you suture the two ends of the bowel together successfully? In large tumors with lymph nodes, distal metastases may be unknowingly present. The most radical procedure is advisable and can only be anatomically accomplished by an abdomino-perineal resection.

These low reconstructions are largely an effort to avoid an abdominal colostomy. Surgical compromise of anatomic excision of all the gland-bearing tissue is an enthusiastic desire to maintain a normal fecal current. Why compromise, for sentimental and esthetic reasons, surgical principles in the treatment of cancer of the rectus? A frank discussion should convince any intelligent person of the advantage of a radical procedure.

An abdominal colostomy properly made, with removal of the primary lesion, can be effectively controlled and will cause a minimum of trouble. The conceptions of a colostomy are based upon inadequate information, sentiment and mental abhorrence. Those who have colostomies properly performed for removable lesions and who have learned a procedure of caring for them have little or no trouble. The bowel in time adapts itself to its new environment and functions with a minimum of inconvenience.

Palliative Colostomy

Palliative colostomy is usually a procedure to be condemned. It gives no palliation and adds little to the patient's life, but contributes much to his discomfort. It is uncontrollable and has contributed much to discredit colostomy performed in association with removable lesions. The pain and discomfort accompanying an unremoved, malignant lesion are attributed to the colostomy rather than to progress of the disease. It is to be performed only when obstruction is present and the lesion is unresectable. It is here only a life saving measure.

In many instances of such advanced states colostomy can be avoided by anastomosing around the lesion and preserving the fecal current, or for the brief period of life remaining, a diet without residue or liquid in character will obviate the annoying effects of a terminal colostomy.

Fixation of Extension

Fixation is often present in colon cancer, and removal may jeopardize surrounding anatomic structures, if caution and care are not exercised in finding a line of cleavage. These structures may be isolated above or below the fixed lesion, followed to the point of involvement and separated with greater certainty of protection. We have found this useful in isolating an adherent ureter or in freeing lesions of the iliac colon from the great vessels.

We have extended the operation of excision in colon and rectal cancer to include many cases which because of fixation and adjacent involvement, we formerly thought, were inoperable and did only a colostomy.

Lesions involving an adjacent loop of small bowel are removed en masse and tubes, ovaries and uterus may be excised along with the cancer if adherent and involved. Rectal lesions involving the posterior wall of the bladder have been extirpated along with a segment of the bladder. The anterior abdominal wall in part has been removed, as have the

surrounding parietal peritoneum and the underlying muscles in lesions of the lateral colon.

In two instances, the left lobe of the liver was removed because of a large isolated metastatic lesion, in the course of the abdominal phase of a low left colon lesion. These extensive procedures are often palliative in nature, but have resulted in an extended and more comfortable life. They have often made unnecessary a palliative colostomy.

Obstruction

Acute obstruction of the colon is reported not to be very common, however, we have had a sufficient number of acute obstructions to form certain opinions and standardize an effective technic for decompression. The frequency of acute colonic obstruction is difficult to determine.

In large clinics where patients are brought from afar the incidence is low, while in congested centers it rises materially. Also an estimation of what an acute obstruction of the colon is, varies. It is sufficient to say that it is a very large dilatation of the colon, sufficient to produce abdominal distress and obvious abdominal distention.

The frequency as reported by Rankin was 5 per cent[1], Gregg and Dixon 5.5 per cent[2], Rea, Smith and Schwyzer 15.2 per cent[3], Campbell 30 per cent plus[4], Burgess 35.6 per cent[5], Graham 16 per cent[6], Scott, White, Brindley (190 cases) 20.6 per cent, University of Minnesota[7], Dennis (367 cases) 9.54 per cent[8]. We have a percentage approaching these reports, there being 12 acute obstructions in 100 cases of cancer of the colon and 8 moderate obstructions, upon which colostomy was done within one to two days after entering the hospital because of progressive obstruction.

Mortality

The operative mortality from surgical decompression of the proximal distended colon is excessive. Brusgaard's report of 13 deaths in 91 cases of volvulus is high, but not excessive, when you consider a volvulus often is devitalized and requires resection.

Brindley reports 7 deaths in 34 cases of acute malignant obstructions. We had 4 in 20 and none in 32 others performed by our present method, using our colostomy clamp. This method is simple, practically aseptic and requires no great hurry in removing the clamp or irrigating the bowel, as it cannot redistend. It is used as a decompression method only for obstruction due to intrinsic cancer, not in acute diverticulitis of the sigmoid.

Colon obstruction is insidious in onset and gradual in progression, except it be a volvulus of the colon. Therefore, it may progress to a marked degree before being suspected, unless one is careful to note the history of progressive obstipation and changes in bowel habits. Too often these patients are treated for a period of time for constipation, spastic colitis, or operated upon for appendicitis. The symptoms are often abdominal in character and not related directly to the obstructive lesion. The clinical manifestations of abdominal colic, forceful, visible peristalsis and borborygmus are present, but in lesser degree than in acute small bowel obstruction. The acuteness of the two lesions are not the same; the symptoms and x-ray findings are different. Vomiting is reflex in character and largely an attempt by nature to empty the stomach and put the intestinal tract at rest. So in like manner the distal segment of the colon may empty itself, as there is nothing wrong with the anatomy and physiology of that segment of the colon. This is more frequently observed in small bowel obstruction and frequently gives a false assurance of security.

The abdomen in a thin individual shows the contour of the distended colon and on the right side the distended cecum can often be outlined. Until acute obstruction actually occurs, often little attempt is made to study the patient and arrive at a diagnosis, though abdominal pain, altered bowel function or possible passage of blood by the rectum are present. It often develops progressively without being suspected until the seriousness of the situation suddenly becomes apparent.

Colon obstructive lesions are of three kinds: malignant, inflammatory and strangulated. It is no serious mistake to err in the immediate differential diagnosis between malignant and inflammatory obstruction, but to do a colon decompression for a volvulus obstruction of the sigmoid is fatal. When doubt exists it is wise to explore the sigmoid by an appropriate incision and if a volvulus is not present to do a right transverse colostomy.

Colon obstruction would seem to be a simple obstruction in which there would be a progressive dilatation of the colon and the small bowel. It is on the contrary, no such simple affair. It soon becomes a closed loop obstruction because of the competency of the ileocecal valve. This is so in two-thirds of the cases and partially so in the remainder. No colon contents regurgitates into the terminal ileum, but, on the contrary, the ileum continues to empty its contents into the cecum. An incompetent ileocecal valve permits some reflux into the ileum and moderate small bowel dilatation. This should not be confusing. The colon gas predominates.

The segment of the colon most distensible is the cecum, the walls of which become extremely thin and ultimately become gangrenous and perforate. This closed loop type of colon obstruction is, therefore, in part comparable to a strangulated or a volvulus type of small bowel obstruction. It is in reality as much of an emergency as small bowel strangulation. Therefore, surgical decompression should not be long deferred, else gangrene and rupture of the cecum will occur. We have observed this twice, and in one instance it occurred pending abdominal preparation for colon decompression. Cecostomy, however, was immediately carried out, and fortunately there was little abdominal soiling

and recovery occurred. Subsequent resection of the lesion was successfully done.

Obstruction presents a complication which prohibits a primary attack upon the lesion, which is quite in contrast to that of small bowel obstruction. In colon obstruction the problem is to decompress the colon and prepare it for subsequent resection of the lesion, while in small bowel obstruction surgery is directed primarily at removal of the lesion. Not only in acute obstruction is decompression desired, but also in all cases of chronic obstruction of the left colon, preliminary proximal bowel decompression is indicated. Only in a clean, unobstructed colon can primary resection and anastomosis be safely performed.

Lesions of the left colon are usually annular, constricting in character and at the time of consultation have a variable degree of obstruction, and a few have complete obstruction. Fortunately, most apparent obstructions are not complete, but are in part due to associated edema, inflammatory reaction and prolapsed mucous membrane. Under proper surgical decompression and by irrigations the obstruction may relent sufficiently to permit the colon to partially empty itself through the distal segment. This materially aids the thorough cleaning of the colon.

Obstructive lesions of the right colon may be decompressed by cecostomy or ileocolostomy. If the obstruction is definite but incomplete, it is quite appropriate to do an ileocolostomy as a preliminary step to right colectomy.

Surgical decompression for lesions more distal can be done by cecostomy or transverse colon colostomy, the latter being preferable if the lesion is low in the left colon. However, in complete obstruction with extensive proximal bowel distention this is not always easy. Often the distended right transverse colon is pushed high under the liver by the enormously distended cecum, which displaces everything well toward the midline and upward, so the transverse colon is difficult to approach and deliver.

Strauss has emphasized the superior value of ileostomy over cecostomy or colostomy in obstruction of the colon and has insisted that the distended colon can be more quickly deflated by passing a colon tube through the ileostomy into the cecum. We have tried this procedure twice and have found it undesirable. The colon cannot be irrigated and cleaned for future surgery. Skin irritation discredits the procedure.

Advocated Procedure for Decompression of the Colon

Through an adequate incision under pentothal or spinal anesthesia (not local anesthesia) we deflate the distended cecum or colon by needle puncture and grasp the deflated bowel at the point of needle puncture with a flat no tooth-thumb forcep. Our special clamp is applied to an adequately suspended cone of bowel. The abdomen is then closed completely around this protruding segment of cecum or colon before it is opened to insert a catheter into the bowel through the round opening in the clamp. The catheter is fixed by suture to prevent its slipping out. It has been pictorially described in other publications (see reference). This procedure does not completely divert the fecal current. It is advocated only for an acute obstruction of the colon due to malignant disease for which resection is subsequently contemplated.

This method exteriorizes only an adequate segment of the cecum or colon and gives a sufficient vent for gas to pass through the catheter. It makes a good skin surface stoma in either instance for irrigation.

The inserted catheter acts as a vent largely for gas and prevents redistention of the bowel. The clamp is removed later, leaving a good stoma and the bowel is later irrigated and cleansed. This method is simple, practically aseptic and is done without difficulty. It always should be done under a light anesthesia. Relaxation and not anxiety and abdominal resistance is very essential for bowel exposure and decompression. We have tried both.

Summary

The anatomical characteristics of the colon are described as they are related to the development of cancer.

The nature of cancer in the various segments of the colon are described.

The more frequent complications as related to the colon segments are discussed.

When possible it is urged to avoid the performance of a palliative colostomy. Its disagreeable features are mentioned.

Abdominal colostomy for removable lesions is advised in certain situations rather than attempted restoration of bowel continuity.

The merits of cecostomy versus colostomy is discussed as a means of decompression of the acutely obstructed colon.

A descriptive discussion of the formation of a surface cecostomy with an abdominal stoma is presented. The technical value of a cecal stoma versus a transverse colon stoma in radical left colon resection is strongly emphasized.

Technical Demonstration of Use of Clamp

Surface Cecostomy as a Procedure for the Decompression of the Acutely Obstructed Colon. Claude J. Hunt, M.D. The American Surgeon. Vol. 20, No. 10, October 1954.

*Clamp is made by V. Mueller & Company, Chicago, Ill.

References

1. Rankin, F. W.: Resection and Obstruction of the Colon (obstructive resection), Surg., Gynec. & Obst. 50: 594-598 (March) 1930.
2. Gregg, R. O., and Dixon, C. F.: Operable Malignant Lesions of the Colon Producing Obstruction, S. Clin. North America. 21: 1143-1152 (Aug.) 1941.
3. Rea, C. E.; Smith, B. A. Jr. and Schwyzer, H.: Carcinoma of Colon and Rectum, Staff Meeting Bulletin Hospitals of the University of Minnesota. 10: 354-364 (April 28) 1939.
4. Campbell, O. J.: Surgery of Carcinoma of the Colon, Minnesota Med. 23: 215-220, (March) 1940.
5. Burgess, A. H.: Treatment of Obstruction of the Colon, Brit. M.J. 2: 584-588, (Oct.) 1934.
6. Graham, R. R.: Carcinoma of the Colon, Am. J. Digest Dis. & Nutrition. 1: 584-588, (Oct.) 1934.
7. Brindley, G. V.: Scott and White. Acute Obstruction of the Colon, Texas State J. Med. 40: 571-577 (March) 1945.
8. Dennis, C.: Treatment of Large Bowel Obstruction. Transverse Colostomy — Incidence of Incompetency of Ileocecal Valve, Experience at University of Minnesota Hospitals, Surgery. 15: 713-734, (May) 1944.

The Care and Planning of Incisions for Surgery of the Breast

Ralph R. Coffey, M.D.

The Educational Program of the American Cancer Society has alerted the public to the importance of early discovery of "lumps" in the breast.

The result of this program has been a moderate increase in the number of women reporting to their doctors for examination of "lumps" they have found by self examination. Many of these cases are clinically benign tumors or cases of cystic disease of the breast. The physician however, cannot, and should not, trust his diagnostic acumen in all cases, and many of these cases require biopsy studies to clarify and confirm the proper diagnosis.

The patient, regardless of age and of the anatomic conformation of this interesting secondary sex organ, is desirous of maintaining its conformation and its pristine beauty.

The surgeon should be interested in conserving this, also, and we present an approach which is old and anatomically correct.

The breast is a highly specialized sebaceous gland lying between the split superficial fascia. There is an anatomical cleavage plane between the posterior layer of the superficial fascia containing the breast, and the deep fascia covering the pectoral muscle. The major blood supply to the breast comes over the antero lateral surface from the thoracic lateral branch of the axillary vessels and over the antero medial surface from the internal mammary collateral vessels. Thus, an incision which allows approach to the breast tissue from its posterior surface avoids the major blood supply and does not disturb the fat which overlies the breast and which gives it a smooth contour.

We have therefore utilized the circum-mammary incision which allows elevation of the breast from the pectoral fascia and bimanual examination to determine if there is more than one suspicious area. After this examination the breast may be everted through the incision and excisional biopsy made of one or more areas, as desired, through the posterior surface.

This approach is attended with minimal blood loss because of full vision and control of the operative area between fingers and thumb. The defects are closed by suture of the posterior superficial fascia and the breast resutured to the chest wall. The contour of the surface of the breast is undisturbed.

A circum areolar incision is occasionally employed in cases of bleeding, or other nipple discharge, and this incision facilitates the identification and isolation of the duct involved, for biopsy.

The areola is put on stretch and "hash" marks made to allow meticulous resuturing of the edges which are handled with "tag" lines or skinhooks.

The fat layer and blood supply is minimal here, if the incision is made inside the circumareolar vein.

Incisions for Radical Mastectomy are planned to take advantage of the segmental and sometome distribution of blood and nerve supply to the skin flaps.

Properly planned the incision allows maximum exposure and access to the axilla and supraclavicular spaces.

The radical incision is planned even before the biopsy incision is made. The patient lies on a sand bag, placed between the scapulae, with the forearm suspended in a sling from the ether shield support and so positioned that the upper arm is at right angles to the body. With this hanging position the neurovascular bundle of the axilla is not overstretched. (Overstretching is one of the causes of postoperative edema of the arm). The lateral end of the incision starts two finger breadths below the skin fold of the arm at the anterior border of the latissimus dorsi. The medial end is on the sternum in the same somotome segment.

The elliptical inclusion of the breast skin and of the skin overlying the tumor can always be adequate, and even generous, and one edge of the elliptical incision follows, in part at least, the biopsy skin incision.

Traction and counter traction on skin edge and breast tissue are done with skin hooks or Lahey Clamps to avoid crushing and compression of breast tissues and skin during the wide dissection of the flaps.

The exposure of the axillary contents and supraclavicular areas is excellent, and closure of this slanting segmental type incision is easily made without tension, or split skin grafts are applied.

Other factors which keep down flap necrosis, aside from careful handling are:

1. Mattress type sutures through the superior flap and below the lower border of the neurovascular bundle in the axilla fashioning a smooth axillary skin with no creases or wrinkles.

2. Suction tubes made from two new 18 French catheters fenestrated and placed at the base of the superior and inferior flaps and exited through stab incisions just medial to the anterior border of the latissimus. Credit is due to A. B. Raffl for this suggestion.

Wangensteen suction through a Y tube evacuates the air and post operative ooze and keeps the flaps adherent to the chest wall with no serum accumulation or wet dressings to encourage bacterial growth.

We now use only one elastoplast adhesive strip over a thin dressing on the incision.

4. Ice bags are kept continuously over the chest wall and axilla for three to four days to keep the skin chilled and decrease oxygen needs of the skin flaps.

Presented at the Annual Meeting, International College of Surgeons, Canadian and United States Sections, Marlborough Hotel, Winnipeg, September 29th, 1960.

5. Varidase Linguets dissolved in the mouth every four hours decrease inflammatory reaction in the traumatized tissue.

6. Arm action is made compulsory in full range several times daily.

7. Wraps of the upper arm are occasionally used. The patients are ambulatory to the bathroom several times a day by clamping the Raffl type tubes.

The increased frequency of biopsies and the consequent psychologic impact on the patient fearing deformity should urge the surgeon to give the patient the best cosmetic result possible.

The circum mammary incision, besides giving a proper surgical approach anatomically, avoiding the major blood supply, allows full exploration of the breast and multiple excisional biopsies with minimal disturbance of breast tissue.

The segmental or somotome type of radical incision allows excellent surgical exposure and dissection and a low lying incision and smooth axillary flap for good cosmetic and functional result. The additional aids to care of the incision and flaps are helpful in obtaining a good end result cosmetically.

Bibliography

1. Gray's Ahatomy, 26th Edition, 1954.
2. Malinac, Jacques: Arterial Blood Supply of the Breast. Archives of Surgery, Vol. 47, p. 329, 1943.
3. Snyder, W. H., Jr., Chaffin, Lawrence: Main Duct Papilloma of the Breast. Archives of Surgery, May, 1955, p. 680.
4. Raffl, A. B.: The use of negative pressure under skin flaps following Radical Mastectomy. Annals of Surgery, 1952, Vol. 136, p. 1048.

Pediatrics

Urosepsis

Harold Davies, M.D.

When a patient at the Children's Hospital is a suspect of having urinary tract infection, a catheterized sample of urine is obtained by aseptic technique, and sent immediately to the bacteriology laboratory. The sample is centrifuged and the sediment examined microscopically. Infection, or Urosepsis, is diagnosed in the laboratory if bacteria and leucocytes are present in this sediment. A culture is then planted for further identification, and in-vitro drug sensitivities.

A preliminary report is sent back, describing the morphology of the organisms, the presence of WBC, and the presence of clumping of WBC, if present.

The instigation of a catheterized specimen for this type of examination may be based on clinical grounds, or upon the finding of an abnormal routine urinalysis.

Although infections at the renal level, usually produce different symptoms than those of the bladder, an absolute localization of the site is not possible or probable. Therefore all cases, including pyelonephritis, pyelitis, cystitis, trigonitis, etc., have been included in this series if they fulfill the above laboratory definition of urosepsis.

Cases of Urosepsis Admitted to Children's Hospital in 1959

There were 58 cases admitted to Children's Hospital, Winnipeg, in 1959 with pyuria and bacilluria, most with urinary symptoms. 28 children were admitted only once, with an acute illness. Recurrent admissions of an acute nature occurred 10 times, and chronic urinary infection with residual damage was discovered in 20 patients.

There were 45 females in the entire group. The age distribution was:

	Patients
Under 1 year	3
Age 1 to 3	13
Age 3 to 5	18
Age over 5	27

The main symptoms were as follows:

	Patients
Fever	27
Frequency	18
Dysuria	13
Pain (in abdomen, back or side)	13
Urinary incontinence	7
Vomiting	6
Enuresis	4
Hematuria	4

The physical examination on admission was reported as negative in 46 instances. Renal tenderness was found in 6 children, and abdominal distention in 2. 4 of the chronic urosepsis had paraplegia and cord bladders. The estimations of blood pressure, hemoglobin and WBC did not aid in the diagnosis, for the most part being all within normal ranges.

Urinalyses were performed on non-centrifuged specimens which were thoroughly mixed. The microscopic examination was done on the high-power, counting 4 fields and averaging. The results follow:

	Cases
WBC — 1 - 10	17
10 - 20	8
20 - 30	10
over 30	21
Clumps of WBC	24
RBC present	8

Albuminuria was reported in 31 of 58 cases, from a trace to 200 mgm., using the Sulfosal A method. Blood urea nitrogen was estimated in 26 cases and found to be over 20 mgm % in 3 only.

The ESR, done on 24 cases, was under 20 in 5 cases, between 20 and 50 in 6, and over 50 in 13 patients with urosepsis.

Urine Cultures

	Cases	
E. coli	40	28 sensitive to Gantrisin, Furadantin, Chloromycetin and the Tetracyclines.
		1 resistant to Furadantin.
		2 resistant to Gantrisin.
		2 resistant to Chloromycetin.
		7 resistant to Tetracyclines.
Proteus	7	sensitive to Gantrisin and Furadantin.
Strep. Fecalis	4	sensitive to Furadantin and Chloromycetin.
Staph. Aureus	4	sensitive to Furadantin and Chloromycetin.
Pseudomonas	2	sensitive to Mandelamine.
Aerobacter	2	sensitive to Furadantin.

I. V. P.

Performed in 43 Cases

	Cases
Found negative (normal)	20
Found dilated calices	9
Found congenital anomaly (duplex kidney - 11)	14
Obstruction uretero-pelvic	1
Single kidney	1
Polyps in ureter	1

Delayed (voiding) Cystogram — performed in 11 cases.
Ureteral reflux found in 5 cases.
No urethral valves were demonstrated on voiding in the boys.

Cystoscopy

Performed in 27 Patients

	Cases
Bladder reported normal	9
Cystitis present	9
Abnormal urethra or bladder neck	13

Retrograde Pyelograms

Performed in 16 patients and further confirmed I.V.P. finding, seldom revealed further pathology.

Surgery

Performed on 15 Patients

Dilatations (urethra and bladder neck)	6
Plastic ureter and pelvis	2
Bladder neck revision	2
Nephrectomy	*1
Suprapubic cystotomy	†4

*Abscess †Neurogenic bladders

A great many cases diagnosed on discharge as acute pyelonephritis were not included in this series. Only one case was included in which a urine culture was not done; and in this case the symptoms were typical, and pyuria with clumps of WBC was found. The rejected cases showed mild pyuria, no clumps, negative urine cultures, and were often admitted as P.U.O.

Failure to culture catheterized urine in a case which is suspect, but shows only mild pyuria, is a serious oversight. 17 cases of mild pyuria fulfilled the necessary requirements in this series for a diagnosis of urosepsis. Pyuria varied considerably from sample to sample.

Similarly bacilluria varied, and a negative culture on a suspect case may not rule out the diagnosis. Some organisms grow poorly in urine and must be sought for repeatedly.

It is interesting to note that, although the textbooks indicate that urosepsis is a disease of the diaper age, the incidence of our hospital admissions was almost entirely over age 1 year. Presumably the difficulty in obtaining a urine sample from an infant, and of obtaining a definite history, makes the diagnosis rare in this group. It would seem logical however, that unless physicians are using antibiotics indiscriminately without a diagnosis, a great many more infants would be hospitalized. The only other conclusion is that the textbooks are misleading in the light of our experience, and urosepsis is a condition of the older child — in this series — over the age of 3 years.

Of the typical cases of urosepsis as we define it, the majority of patients were female, but only 3 to 1. Males were subject to acute infection and chronic, but not recurrent. Fever occurred in less than half, but urinary symptoms occurred in most patients. E. coli was found to be the infecting organism in 69% of this series. Furadantin appeared to be the most effective drug in the vitro testing of sensitivity, but Gantrisin and Chloromycetin were much the same. Furadantin would seem the most effective, if the other organisms found are also included. Because of the possibility of finding a resistant organism, cultures and sensitivity tests are mandatory.

The presence of hematuria and albuminuria was inconsistent and did not aid in the diagnosis, nor in the evaluation of any complicating factors.

An intravenous pyelogram was performed on 43 cases. The presence of an anomaly in one-third of these, testifies to the value of the test, and to the significance of a congenital abnormality in urosepsis. The question that a duplex kidney has any relationship to urinary infection has been raised. The presence of 11 such kidneys in 43 patients would seem more than accidental. However, series of autopsies on children report 5 - 12% incidence of anomalies of the urinary tract. It would seem essential in the evaluation of a patient with urosepsis to have I.V.P. studies.

If an abnormal kidney or ureter is found, then the relationships with the sepsis should be considered. A deformity which interferes, even in the slightest way, with urinary drainage may be a contributing factor. Also if one congenital anomaly is discovered, a thorough search for another should follow.

Ureteral reflux has been discussed in the literature as the mechanism for the ascension of bacilli from the bladder to the kidney. Delayed or voiding cystograms have been the methods employed to check for this phenomenon. It was performed in 11 patients and found positive in 5, only one bilaterally. Reflux[1, 4] possibly indicates some urinary obstructive element at the bladder level or lower. Its significance has not been fully evaluated.

Cystoscopy was performed in 27 patients, either with recurrent or chronic urosepsis. An abnormal urethra or bladder neck was reported in 13 patients. If the 4 cases of neurogenic bladder are removed from the total of the recurrent and chronic, there would be 26. One-half of these had urethral valves or folds, or bladder neck folds or hypertrophy. This would appear highly significant as to cause and management of chronic and recurrent urosepsis.

Discussion

Urosepsis has been chosen as a term to signify infection in the urinary tract, rather than cystitis and pyelitis, because it has been recognized that the majority of cases represent an ascending infection which involves the whole urinary tract.

Improperly handled patients may be left with serious residual damage. Congenital and acquired defects contribute the underlying etiological factor to those with recurrent and chronic urosepsis. The demonstration of abnormalities by radiological techniques has improved in recent years. Now we can request improved intravenous pyelograms, voiding and delayed cystograms, and floating lipiodol for residual urine[3]. Pryles and Steg[2] have compared methods for bacteriologic diagnosis, when catheterization is undesirable. They state that urinary sepsis is present if the colony count is over 100,000 per c.c. in clean voided specimens. Members of the staff at Children's Hospital were asked for comments:

Bacteriologist — The method of Pryles and Steg of colony counting in voided specimens is not difficult. Its value is lost unless the technique of collection is very rigid, and the sample delivered immediately to the laboratory.

Paediatrician — Single acute urinary sepsis is not uncommon and probably does not require radiological investigation. Many infants, suspect pyelonephritis, are treated with sulfas with such good results that nothing further is carried out. Recurrent urosepsis, without demonstrable X-ray or cystoscopic abnormalities, may require prolonged drug therapy for several months.

Radiologist — Newer contrast media and techniques give excellent results in demonstrating abnormalities. The method of delayed cystograms often gives unusual findings; in one case, a large reflux to a dilated pelvis after waiting for an hour. Voiding urethrograms have been done at the same time in all boys, looking for valves.

Urologist—The current experience seems divided as to the significance of ureteral reflux; as to whether it represents a lower urinary obstruction, an abnormal uretero-vesicular insertion, or a normal function. A number of children have been found with narrow bladder necks, which may be the cause of relative bladder incompetence and residual urine. This may progress to chronic infection and damage to ureters and kidneys. Successful revision operations of the bladder neck have been performed, although dilatations often suffice.

The purpose of this paper is to bring attention to the clinical aspects of urosepsis, and the current methods of investigation and treatment of the chronic or recurring cases.

References

1. Vesico-ureteral Reflux in Children: Pasquier et al. South M.J., 52: 967, 1959.
2. Specimens of Urine by Catheter versus Voiding: Pryles and Steg. Pediatrics, 23: 441, 1959.
3. Floating Lipiodol: Roberts and Latimer. J. Ped., 54: 68, 1959.
5. Persistent and Recurrent Urinary Infection in Children: Forsythe and Wallace. Br. J. Urol., 30: 297, 1958.

. . . Although the American doctor can do his rounds in a Mercury and send his wife shopping in a mink, his patients give him a longer run for his money than in Britain. Ill-health is as much a national preoccupation in both countries, but the American is exposed to many more diseases—from one copy of The Reader's Digest alone you can catch anything from schizophrenia to shingles. And the obituary pages of the papers give everyone's reason for appearing in them with such rich detail they leave their readers with one finger on their pulse and the other on their doctor's doorbell. The American hypochondriac is far better educated in scientific polysyllables than our own, who generally has as little idea of what goes on inside his thorax as inside Harwell, and he opens the consultation with the assertion "I gotta split P-wave in my lead three, betcher a million dollars!" This can sometimes be awkward for the doctor, unless he's mugged up the same issue of Time.

This necessity of keeping one disease ahead of the patient perhaps accounts for the American doctor looking like a doctor. The British doctor floundering about with the bedclothes, wondering where the devil he can wash his hands, and trying to light his 1935 auroscope, has a horror of dressing up, even for surgery, and our most formal specialists only succeed in resembling ordinary barristers. But the most insignificant of American physicians appears in white trousers and jacket buttoned to the neck, presenting an appearance of sterility unseen in this country outside West End gentlemen's hairdressers, and is widely pictured thus in advertisements recommending such potentially lethal articles as motor-cars and cigarettes. . .

—PUNCH, November 20, 1957.

Orthopaedics

Orthopaedic Aspects of Reiter's Syndrome

P. N. Porritt
F.R.C.S. (Eng.), (C), L.R.C.P. (Lond.), (C)

Reiter's syndrome, as first described in 1916 by Reiter, comprises urethritis, polyarthritis and ocular inflammation. Since the war, investigators in London and Scandinavia have recognized imcomplete and chronic forms of the syndrome. In this paper I propose to discuss the orthopaedic aspects of two forms,

1) Acute onset
2) Insidious onset.

Etiology

Age incidence: Most commonly the syndrome occurs between 20 and 40 years of age, but Paronen has described a case in a boy 2 years and 8 months old.

Sex incidence: Much commoner in males. In Csonka's series of 185 patients there were only 3 females, and Paronen found that 10% of his 334 cases were females.

The cause is unknown.

The syndrome may follow: 1) An attack of dysentery, 2) Genital infection.

Reiter's case followed an attack of Shiga dysentery and in his investigations found a spirochaete, which was regarded as the causative organism. Spirochaetes have never been isolated again.

In 1948 Paronen described 334 cases, which developed 10 to 100 days after an attack of Flexner dysentery.

In most other series, however, there was no evidence of dysentery, but there was evidence of genital infection, mostly after sexual relationships. (Csonka, Oates, Ford).

It has been established that gonorrhoea is not related to this syndrome, but a non-specific genital infection is acquired simultaneously.

In 1942, Dienes et al isolated a pleuro-pneumonia like organism, which was regarded as the causative organism for some years. However, recent work has not confirmed this. (Csonka and Ford).

Due to considerable similarity to rheumatoid arthritis and ankylosing spondylitis, it has been suggested that Reiter's syndrome belongs to the Collagen group of disorders.

Clinical Forms

1. Acute Onset

Usually this variety manifests itself 3 - 10 days after an attack of dysentery or urethritis, but in exceptional cases there may be a latent period up to 100 days.

The complete syndrome comprises:

Urethritis
Conjunctivitis, Iritis, or Uveitis
Polyarthritis.

Other manifestations may include skin lesions, such as keratodermia blenorrhagica; myocarditis or pleurisy.

The patient usually has a fever and malaise.

The joints are swollen, warm and painful to move; the overlying skin is not erythematous. The feet, ankle, knee, elbow and wrist joints may be involved, but the hands and fingers are not usually affected.

There may be low back pain possibly due to acute sacroiliitis, and plantar fasciitis occurs in about 20% of cases.

The full triad is present in the majority of cases. It must be emphasized that: a) One or other component of the triad may not develop. b) That all 3 elements may occur at different times.

Case Report — Patient of Dr. Michael Newman

A 28-year-old male was first seen on January 26, 1960, complaining of four days low back pain, frequency of micturition and terminal hematuria. There was a history of recent sexual intercourse. On examination he had a temperature of 100. There was a profuse mucoid urethral discharge, tender prostate and iritis. Laboratory examination confirmed the clinical diagnosis of gonorrhoea.

The gonorrhoea was treated with penicillin, the iritis with terramycin eye drops. On January 28th he developed pain in the right hip. On January 31st he developed evidence of polyarthritis with involvement of the left hip, left ankle and subsidence of pain in the right hip. During the course of the next ten days he subsequently developed involvement of both knees, right wrist and right foot. He was treated initially with Butazolidin and aspirin, but as the disease became more progressive on March 2nd a course of Prednisone was begun which continued until May 12th. The symptoms subsided in the joints by March 24th. There was some residual effusion in both knees and the right ankle until April 7th.

Comment: This man illustrates the full syndrome, but the urethritis and iritis preceded the polyarthritis.

Prognosis

80% of Paronen's cases became asymptomatic within sixteen months, the majority within a few months. 20% had mild joint discomfort and swelling for about 3 years when the follow-up ceased.

Csonka was able to follow some of his patients for many years and found:

a) 104 of 185 patients had recurrent attacks of polyarthritis — averaging about 3 attacks. One man had 4 attacks in 25 years.

b) Only 23 had permanent joint changes, and these were similar to those of rheumatoid arthritis: destruction of joint surfaces, deformity, and in a few, ankylosis.

2. Insidious Onset

These patients slowly develop pain and swelling in one joint, gradually other joints become involved and so they appear to be suffering from rheumatoid arthritis.

The patients are in good general health and not anemic. There may be a past history of eye changes or urethritis.

The mode of presentation may be: a) Arthritis of ankle, tarsal or metatarsophalangeal joint, or major upper limb joint. b) Plantar fasciitis. c) Low backache.

The diagnosis is made by: a) Distribution of arthritis which nearly always involves the feet and seldom the hands. b) History of presence of eye involvement. c) Evidence of genital infection — notably chronic prostatitis. d) X-ray evidence of joint changes — erosions of the articular surface and subjacent sclerosis leading ultimately to destruction of joint surfaces. There is often a fluffy periostitis on the posterior or postero-inferior aspects of the os calcis with spur formation. e) Latex fixation test — consistently negative. f) In a fit man without anemia.

Case Report No. 2

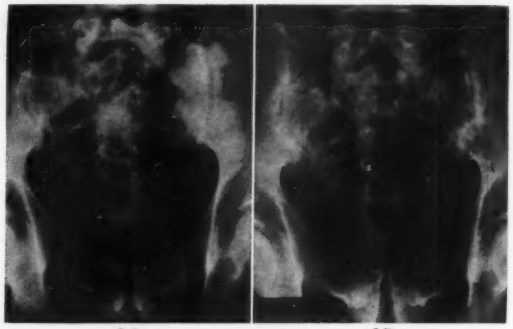
A male, now aged 33, who is a part time vagrant, is known to have had two attacks of gonorrhoea, one of which was in 1948. In 1955 he was diagnosed as having ankylosing spondylitis (Figure 1). He was treated with deep x-ray therapy with temporary relief of low back pain. In 1957 he developed pain and swelling at the base of the fifth right metatarsal and over the left tibial tuberosity. X-rays of the base of the fifth metatarsal showed some erosion with no new bone formation (Figure 2A). X-ray of the left tibial tuberosity showed no abnormality (Figure 3A). He was seen again in 1958 with persistent pain in these two sites, and x-rays showed further evidence of destruction of the base of the first metatarsal (Figure 2B) and evidence of an erosive area of the tibial tuberosity (Figure 3B). During the period under observation he had recurrent attacks of conjunctivitis, lasting up to 2-3 weeks. Prostatistic massage revealed evidence of chronic prostatitis.

Case Report No. 3

A male, aged 21, at the onset of his disease in 1951, commenced with a bilateral plantar fasciitis, x-rays of the os calcis at that time showing no abnormalities. A few months later he developed arthritis of all the metatarsophalangeal joints of both feet. Again x-ray examination revealed no abnormality. In 1952 he developed pain in the sacroiliac area lasting for a few weeks only. X-ray showed no abnormality.

Progress of the Disease

He has had intermittent plantar fasciitis with radiological evidence of development of huge plantar spurs similar to those shown in Figure 4. He had persistent pain in the metatarsophalangeal

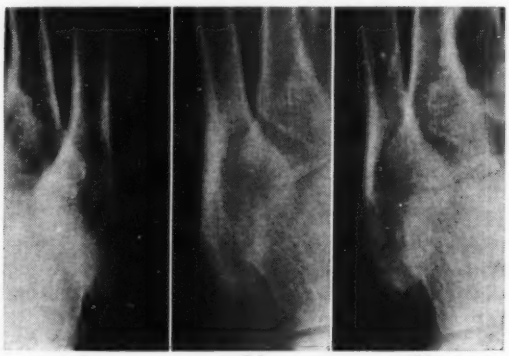


1A 1B

Figure 1

1A: X-ray of Sacroiliac Joints in 1955, showing early evidence of ankylosing spondylitis and early sclerosis of the symphysis pubis.

1B: Shows changes in 1958 with marked erosion and new bone formation of the symphysis pubis and further destruction of the left sacroiliac joint.



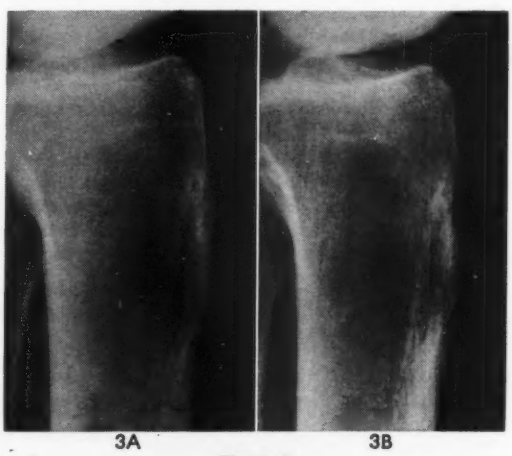
2 2A 2B

Figure 2

2: Control.

2A: 1957, showing early destructive changes at the base of the fifth metatarsal.

2B: 1958, showing further destructive changes.



3A 3B

Figure 3

3A: X-ray showing tibial tuberosity in 1957 which appears normal.

3B: X-ray in 1958 showing erosion of the superior part of the tibial tuberosity.

joints, radiological evidence of progressive destruction of mainly the heads of the metatarsals and to a lesser extent of the bases of the proximal phalanges. There was no further low back pain, but serial x-rays of the sacroiliac joint showed progressive destruction and obliteration of the sacroiliac joints producing an appearance identical with that of ankylosing spondylitis. No other joints were involved either clinically or radiologically. He was found to have evidence of chronic prostatitis, but there had never been any involvement of the eyes.

Case Report No. 4

Male, aged 32, in 1960. Since the age of 14 he had had intermittent attacks of pain which had become more persistent during the past five years involving: 1) Metatarsophalangeal joints. 2) Sacroiliac joint region. 3) Right shoulder. 4) Both heels, in the form of plantar fasciitis.

X-rays of the heels show massive fluffy plantar spurs (Figure 4). There are very early destructive changes in the left sacroiliac joint, the right being normal at present. X-rays of the hand revealed no abnormality. There was no conclusive evidence of chronic prostatitis.

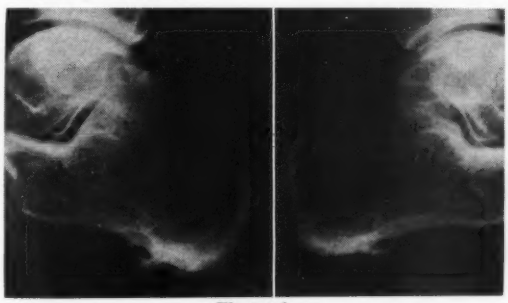


Figure 4

Fig. 4: X-rays of os calcis showing massive fluffy plantar spurs.

Comment

All three patients have sedimentation rates in the region of 30 mm. in one hour, negative latex fixation test and negative serological tests for syphilis and gonorrhoea.

These three cases illustrate three possible variants of the chronic form. Case No. 2 presents all three components of the triad, Case No. 3 has never had any involvement of the eyes and Case No. 4 has only polyarthritis. Case No. 4 could be regarded as a rheumatoid arthritis, but the unusual distribution of his polyarthritis without involvement of the hands, early changes in the sacroiliac joints, and the persistently negative latex fixation test are against the diagnosis.

Discussion

Since the last war a considerable number of papers have been published and two major approaches have been:

1. Definition of complete and incomplete forms of Reiter's syndrome and the differentiation from ankylosing spondylitis and rheumatoid arthritis.

2. Attempts to decide the nature and cause of Reiter's syndrome.

Chart Number 1 illustrates differentiating points between Reiter's syndrome, ankylosing spondylitis and rheumatoid arthritis. It will be seen that ankylosing spondylitis and Reiter's syndrome are diseases of males predominantly, whereas rheumatoid arthritis involves females predominantly. The distribution of joint involvement is significantly different in all three forms. Reiter's disease involving mainly the foot and ankle joints and very rarely the hands; ankylosing spondylitis affects the spine; while rheumatoid arthritis affects the hands and to a less extent the feet. The latex fixation test is persistently negative in patients with ankylosing spondylitis and Reiter's disease whereas it is positive in 30% or more of patients with rheumatoid arthritis.

There is evidence of chronic prostatitis in a very high percentage of patients with Reiter's syndrome and ankylosing spondylitis, but only 30% in patients with rheumatoid arthritis and in a normal control series.

It can be seen from the chart that the differences between these three conditions are clear and there seems no logical reason for considering ankylosing spondylitis a form of rheumatoid arthritis.

	REITER'S SYNDROME	ANKYLOSING SPONDYLITIS	RHEUMATOID ARTHRITIS
AGE	20 - 40	20 - 40	30 - 50
SEX	♂	♂	♀/♂ 3 / 1
SKIN NODULE	O	VERY RARE	COMMON
PERIPHERAL JOINT LESIONS	FEET - USUAL KNEES - ELBOW HANDS - RARE	FEET KNEE - ELBOW } RARE HANDS	HANDS - USUAL KNEE - ELBOW FEET COMMON
SACRO - ILIITIS	COMMON	100%	10%
PROSTATITIS	95%	80%	30%
LATEX FIXATION TEST	- VE	- VE	+ VE IN 30% +

Chart I

Shows the differentiating features between Reiter's Syndrome, Ankylosing Spondylitis, and Rheumatoid Arthritis.

Nature and Cause of Reiter's Syndrome

This remains unsolved. No causative organism has been isolated.

Histological examination of synovial tissues affected has not yet been of assistance. Inge states that the histological pattern of most varieties of chronic synovitis (rheumatoid, infective or traumatic) is the same. Further investigation of the histological appearance is indicated, as very little work has been attempted in this field.

Treatment

No therapy recorded in the literature has been of avail.

As it has been found that the symptoms of non-specific urethritis have responded to a short course of one of the tetracyclines, I have tried a course for six weeks in six patients.

Results

Three are symptom-free.
One considerably improved.
One benefited temporarily.
One is not improved.

It must be emphasized that the method of treatment is purely empirical and is still experimental. Many cases of Reiter's disease have symptom-free periods lasting many years before subsequent attacks develop. Until the patients have been followed for another ten years at least it will be impossible to be certain that the three who are symptom-free have been cured.

Conclusion

The problems to be solved are the nature, cause, treatment of this condition, but to be able to attack these problems the condition must be recognized first. Almost certainly many cases of Reiter's disease are not being diagnosed.

Acknowledgments

I wish to acknowledge my gratitude to Mr. A. R. Gibson at Children's Hospital, for the photography of the illustrations.

I am grateful to Dr. Michael Newman for permission to publish the details of Case No. 1.

References

1. Boyd, Csonka, G. W. and Oates, J. K.: Epidemiology of Non-specific Urethritis. Brit. J. Vener. Dis. (1958), 30-40.
2. Csonka, G. W.: Laboratory Investigation in Reiter's Disease. Brit. J. Vener. Dis. (1959), 34-84.
3. Csonka, G. W.: Reiter's syndrome. B.M.J., 10: 5, 58.
4. Ford, D. K.: Tissue Studies in Non-specific Urethritis and Reiter's Syndrome. Brit. J. Vener. Dis. 1958), 34-53.
5. Gartmann: Studies in Prostatitis. Brit. J. Vener. Dis. (1958), 34-181.
6. Grainger, R. G. and Nicol, C. S.: Pelvic Infection as a Cause of Bilateral Sacroiliac Arthritis and Ankylosing Spondylitis. Brit. J. Vener. Dis. (1959), 35-92.
7. Mason, R. M.: Ankylosing Spondylitis. Brit. J. Vener. Dis. (1959), 35-71.
8. Mason, R. M., Murray, R. S., Oates, J. K., Young, A. C.: Prostatitis and Ankylosing Spondylitis. B.M.J. (1958), 1-748.
9. Oates, J. K.: Sacroileitis in Reiter's Disease. Brit. J. Vener. Dis. (1958), 34-177.
10. Oates, J. K.: Diagnosis of Chronic Prostatitis. Brit. J. Vener. Dis. (1958), 34-250.
11. Oates, J. K.: Incidence of Genital Infection in Males with Ankylosing Spondylitis. Brit. J. Vener. Dis. (1959), 35-89.
12. Paronen: Acta Med. Scand. 131, Suppl. 212, (1958).

Health League of Canada

The Health League of Canada Sponsors the 17th National Health Week, March 12th to 18th, 1961

The Health League of Canada was organized in 1919, as a voluntary organization, national in scope, and dedicated to raising the standards of living in this country by raising the standards of health.

It has been long established that the greatest single cause of poverty is illness. Most illness may be prevented, and it is the prevention of illness that is the goal of the league.

The League attempts to reach the very heart of the country — every Canadian family. This is done through Health Magazine, published every two months by the League, through National Health Week every February, and through National Immunization Week each Fall.

The press, radio and television receive authoritative material on health matters regularly throughout the year from the League headquarters in Toronto, which also distributes films, publishes pamphlets, arranges public meetings and other aids, leading to the fullest possible dissemination of up-to-date information on public health.

All fields of health are covered by the League, specifically: child and maternal health, nutrition, foodhandling, pasteurization of milk, fluoridation of water, social hygiene, immunization, gerontology, artificial respiration, and industrial health.

Scholarship Awards

To help provide specialized training for persons working with crippled children and handicapped adults, fellowship and scholarship awards are available from the National Society for Crippled Children and Adults.

Twenty fellowships to professional workers for specialized training in counselling and job placement of cerebral palsied persons are financed by Alpha Gamma Delta, international women's fraternity, and administered by the National Society.

Scholarships, co-sponsored by the National Society and Alpha Chi Omega, national women's fraternity, are available to help supplement and increase the skill and knowledge of professional personnel in serving the cerebral palsied.

March 15, 1961, is the deadline for filing fellowship applications. These awards are available to counsellors, employment interviewers, placement personnel and other professionally qualified persons working with the handicapped whose responsibility include vocational counselling and job placement.

Fellowships cover four weeks of specialized training, June 12 - July 7, 1961, at the Institute of Physical Medicine and Rehabilitation at New York University Medical Center. Awards in the amount of $300 will cover tuition and laboratory fees and a moderate amount of living expenses. The course will include lectures and demonstrations on all phases of physical rehabilitation, with instruction provided by members of the institute staff and other specialists in this field.

The deadline for filing scholarship applications is April 1, 1961. These awards are available to professional personnel including orthopedic surgeons, neurologists, pediatricians, psychiatrists, and other medical specialties; physical and occupational therapists; speech pathologists and therapists; and others who work with the crippled such as educators, including nursery school teachers and teachers of special education, psychologists, social workers and nurses.

Scholarship grants are given in varying amounts up to $750 depending upon the length and scope of the course and the availability of other financial aid. Courses for which scholarships are granted must have primary emphasis on cerebral palsy and participation of a member of the American Academy for Cerebral Palsy or have the prior approval of members of this Academy serving as counsellors to the National Society for Crippled Children and adults.

Full particulars and application forms on these fellowship and scholarship awards can be secured from the Personnel Service of the National Society for Crippled Children and Adults, 2023 West Ogden Ave., Chicago 12, Ill.

The Organized Charity, Scrimped and Iced

J. H. Lindsay, M.D.

"There is a time in every man's education when he arrives at the conviction that envy is ignorance; that imitation is suicide; that he must take himself for better, for worse, as his portion: that, though the wide universe is full of good, no kernel of nourishing corn can come to him but through his toil bestowed on that plot of ground which is given him to till . . ."

"Society everywhere is in conspiracy against the manhood of every one of its members . . ."

"I am ashamed to think how easily we capitulate to badges and names, to large societies and dead institutions . . ."

"I tell thee, thou foolish philanthropist, that I grudge the dollar, the dime, the cent I give to such men as do not belong to me, and to whom I do not belong . . . the education at college of fools; the building of meeting houses to the vain end to which many now stand; alms to sots; and the thousandfold Relief Societies . . . though I confess with shame I sometimes succumb and give the dollar, it is a wicked dollar which by and by I shall have the manhood to withhold."

"Go, love thy infant, love thy wood-chopper, be good-natured and modest . . . thy love afar is spite at home."

Ralph Waldo Emerson would be considered as a non-conforming isolationist, if he expressed the above comments today, rather than in 1841 when he wrote his essay, Self-Reliance.

When TV tells us in no subliminal terms to give in the community way and when I receive frequent requests to help build more halls of higher learning, and learn by daily contacts how our over-solicitous governing bodies are robbing people of the opportunity to gain self-respect by self-accomplishment I wonder if his philosophy should not have influenced our educators a bit more.

Mental ill health is receiving much attention today, and many well-meaning endeavors are being made to correct it. My daily efforts are certainly well-meaning and as precise as I can make them to correct actual disease or relieve the anxieties that result from a way of life that does not give a sense of accomplishment or satisfaction. Individual or group efforts to relieve these increasing anxieties, even with the dubious aid of tranquillizers and injurious self-medication with alcohol by increasing thousands, is rather futile and discouraging except to a blind enthusiast. I wonder at times if some of our efforts are not only futile but whether they are perpetuating and increasing the problem of anxiety and tension that results from unhealthy attitudes.

It is comforting as well as enervating, to know that our legislators have yielded to public pressure and provide "full coverage" for us from foetus to ashes. But we should not blame our governments for doing what we demand of them. We must instead look to ourselves and ask where and when we lost the courage to be self-reliant, at least to the tiny degree that we could retain some self-respect which is the only sound foundation for mental health.

Perhaps our democratic way of life is like the witch that almost enticed Hansel and Gretel to their doom with the promise of sweets. Certainly, if its promise of "Freedom" entices large groups of our society to let their children suffer while they pursue it there must be some reason for mild misgivings at least.

Is this perhaps a capitulation to badges and names that Emerson was ashamed of 100 years ago? Our capitulation today appears to be hastened by the propagandizing we are subjected to along with our entertainment, but very few are objecting, it appears. The appeals are answered dutifully as good members of our society should, but the satisfaction is a very dilute and impersonal one. Perhaps this expression of "love afar" does not cause "spite at home" as Emerson said, but neither does it in many cases give the unknown recipient a warm feeling of gratitude.

Perhaps Emerson knew that the recipient of the charity dollar in his day would lose more in self-respect than he gained materially, and that this practice of his foolish philanthropist, if prolonged over several generations, would create the social problems of today — problems characteristic of a welfare state where people are reacting to an abnormal and shaming dependence by normal irritation and aggression. In other words, are they like a drowning man fighting desperately to avoid complete passivity and death?

Perhaps, we should ask ourselves whether our smothering type of widespread philanthropy should be modified rather drastically, to allow our community millions to be diverted from what appears to be their debasing effect on self-respect to improving elementary teaching of our children. Competitive attitudes for self gain are both homicidal and suicidal today. They do give an over-all kind of self-satisfaction to those who are strong, but only a small minority have that kind of strength. These competitive attitudes are basic in our present teaching system which it appears has taught us poorly to deal with personal, community and international problems.

Our dollars appear to be acting like the sorcerer's apprentice, merely perpetuating a problem we are becoming frantic about. That of giving more and more help to more and more people who are degraded by clamouring for it. Whether our society

could weather the temporary storm of protest that would precede a change-over to a constructive educational approach I don't know. It would take considerable scurrying around to find philosophers capable of imparting a new set of courageous, co-operative attitudes to teachers for our children's children. Who would keep a hungry mob of competitive, suspicious parents at bay while this was being accomplished I don't quite know. Perhaps we should do an extensive job of fumigating unless some superhuman humanitarian did appear, as in the movies, and threatened to exterminate us if we didn't stop our squealing and squabbling in both high and low places.

Once started on the Utopian educational scheme we could stop pouring good money after bad. We would perhaps realize that society as we have made it recently is really a demoralizing charity institute and not the fine Welfare State we are still trying to delude ourselves into believing it is. "The organized charity, scrimped and iced in the name of a cautious statistical Christ" has very little real chance of answering our needs to correct the social miseries we are constantly talking about. The proposed puny penal reforms are admirable indeed, but are not going to correct the faulty ways of thinking that make crime attractive to many people. Attempts at correction by offering more comfortable periods of incarceration will not antagonize the law breaker as much and it will make his family happier on their week-end visits, but in many cases will simply increase the contempt that the criminal already feels for our society.

Our various welfare agencies are also admirable in their noble humanitarian efforts, but their efforts are years too late to really help their "clients." It is true that they dispense a great deal of comfort to the needy, but the needy are beginning to outnumber the givers and, often with the help of vociferous organizers, beginning to demand as their right what started out as charity packages, subsidies and various kinds of insurance and "fringe benefits."

But to return to a Utopian dream state, if we could carefully select and train teachers as we do our scientists and soldiers, we should eventually have a group of people who could teach children what a few philosophers and poets have always cried from their ivory towers in the wilderness — a reverence for Life, a trusting courageous acceptance of the opportunity to do their particular job in the world, rather than a resentful contemptuous attitude toward a society that preaches equalities and freedoms that are quite impossibly incompatible with our way of life. It is this confusing inconsistency that so many bewildered children feel when they "come out" and are presented to society. Some of the fortunate ones are able to "adjust" to a generalized maladjustment and thus perpetuate the system — many others however require the help of "mental illness" or beatnik groups or the pleasant notoriety of crime to give their life a satisfactory meaning or set of values. We apparently have to stop subsidizing bitter-sweet charities, top-heavy, top-level talks and begin subsidizing a group of Christian non-conformists. Let's see what they can do at teaching our children with the same efficiency at the bottom level as our conformist social atomizers are using at the top levels.

A Utopian dream to be sure, but better than the nightmare of trying to supply more and more social workers, psychiatrists, penologists, sicknick humorists, "fringe benefits" and bombs to relieve the miseries of our scrimped and iced charity society.

Too many defence, charity, welfare and non-correctional justice dollars are available and too pitifully few educational dollars. Before we start on this enlightened course, however, we must protect ourselves from hungry hoards of unenlightened barbarians so perhaps we have made a full circle and come back to where we started, facing animal destructive tendencies and unable to see above them. But let's keep trying to correct man's inhumanity to man and let's try some drastically new educational methods.

P.S. — The ingenious gentleman Don Quixote is my favourite commentator on world events. — L.

Keep Pace With Medical Progress

March 27 - 30 — Vancouver

This Fifth Annual Scientific Assembly is planned by the College of General Practice to keep general practitioners abreast of those medical developments which are of particular concern in the field of general medicine. The programme will warrant 14 formal study credits for College members but non members will benefit equally by attendance at these sessions.

20 Medical Authorities from Scotland, Australia, Canada and United States in addresses and round table discussions.

More than 100 technical and scientific exhibits.

Medical films of special interest to general practitioners.

A full Ladies' programme to engage doctors' wives during all four days.

Editorial

S. Vaisrub, M.D., M.R.C.P. (Lond.), F.R.C.P. (C.), F.A.C.P., Editor

Pronoia and the Brave World

"Diagnosis at dusk," a satire on contemporary medical mores published in 1954 and reprinted in this issue, can hardly be called a literary masterpiece. A product of a mind obviously immature and lacking in subtlety, it offers but little amusement to the discriminating reader. Nonetheless, we have acceded to the request for its republication (see "Letter to the Editor"), if only to recall attention to its theme — the displacement of the traditional clinical approach by indiscriminate laboratory investigation.

The unbounded enthusiasm with which we embrace every new laboratory test and every new diagnostic tool, is a relatively recent phenomenon. Our medical forbears, reluctant to clasp anything new to their bosoms, stoutly resisted every instrumental innovation. Even the stethoscope, one of the pillars of Physical Diagnosis, failed to gain immediate acceptance when it was first introduced by Laennec in 1815. In fact, it was derided by most of the influential clinicians, and denounced by the Royal College of Physicians as a menace to "clinical medicine" which brooks no artificial devices to aid the auscultating ear.

This conflict between the clinical and instrumental, so apparent and at times so dramatic in the history of Medicine, has doubtless many counterparts in other fields of human endeavour. It would be interesting to speculate on how the tribal chieftains of the stone age welcomed the invention of the bow and arrow, or how the members of ancient glee clubs greeted the introduction of the first wind instrument by Tubal Cain. Did they too see in these instrumental intrusions a menace to "clinical" warfare or "clinical" music?

The picture has now changed radically. Just as new weapons for arsenals and new instruments for orchestras, so new apparatus for the laboratories are welcome arrivals, and no one but an inveterate sentimentalist would long for the return of the good old days. Many, however, fear that preoccupation with the instrumental will lead to the neglect of the clinical. "Dialogue of tomorrow" (Lancet 1: 7/32: 1022 May 7/60) records the following conversation of the future:

> "Sir, can you tell me the meaning of an obsolete word, I know medical history is a hobby of yours.
> I'll try, what word?
> Auscultation.
> It meant listening.
> Listening to what, Sir?
> Sounds made by the human body. Doctors used to listen to their patients' bodies and palpate them too."

Exaggerated as these projections into the future may seem, they are not without historic precedent. Uroscopy, widely practiced in the middle ages completely dispensed with physical examination. Diagnosis was made by inspection — contemplation is perhaps the better word — not of the patient, but of his carefully bottled urine. Pronoia, one step removed from uroscopy, has disdainfully eliminated not only the physical examination, but also the anamnesis. Without examining or questioning the patient the physician practicing Pronoia arrived at diagnosis and therapy by "intuition."

May we not look forward to modern analogues of Uroscopy and Pronoia when things get "real tough," and powers beyond the physician's control throw into his lap a patient load which he will be unable to handle? Think of the vast numbers of patients he will be able to "see" after he has eliminated the history and the physical examination. With multitudes of patients handled by "intuition" and countless reports—by "automation," he will have the Time problem licked in no time.

1984 or may be sooner.

Ed.

☆

Letter To The Editor

Thumbing through the bound volume of the Manitoba Medical Review circa 1954 (yes, I bind them, heaven only knows why) I came across an offbeat satire titled "Diagnosis at Dusk" which I deem worth reprinting. Although somewhat puerile — it carries a message which cannot be repeated too often.

M. Y. Alterego.

Editor's Note:

We cannot agree more. The satire is reprinted, (vide infra) and commented upon in the editorial (vide supra).

☆

Diagnosis at Dusk

I was on the spot. Six p.m. and no diagnosis. It had to be made and it had to be made fast. I was thinking hard. If I only knew where to begin. Then it hit me. Electrocardiogram, of course! I pressed the buzzer. Marilyn came in, brisk and beautiful, an eyeful of femininity. But I was in no mood for inspection. "The E.C.G.," I barked, "be quick!" Accustomed to lightning action she delved into the right drawer and handed me the tracing. There was no time to waste. Quickly I glanced over the limb leads and the unipolars. Nothing here. Ah, the esophageal! Staring at me was a tantalizing T wave, not upright, not inverted, just flat.

What do I make of it? Nothing definite. Borderline. I threw the record into the waste basket and rang again.

Marilyn breezed in reeking of Chanel 5. But I was not in the mood for Olfaction, "The X-rays," I roared. Instantly she produced. The films were still wet and dripping. But I was past all caution. I snatched them from her hand and held them up to the Viewer. The chest-heavy roots, tied diaphragms, unfolded aorta. Routine stuff! Barium series—deformed cap? Not quite. Filling defect? Not b . . . likely. The contrast enema — a small diverticulum. Or is it a fecolith? As if it matters! The intravenous pyelogram — are the calyces clubbed? Not much. The retrograde pyelogram—disgustingly normal. The K.U.B. and gall bladder visualization even more so. No help here. I threw the films out the window. I rang again.

Marilyn waltzed in wearing a red tight-fitted sweater. But I was not in the mood for palpation. I was faced with a dilemma. Blood or urine? Quickly I made a decision. "Blood chemistry," I shouted. Promptly she brought in the file. Glucose tolerance curve—flat? Not too significant. B.U.N. —upper limit of normal. Urea clearance — lower border of normal. Serum sodium, serum potassium, serum calcium, serum chloride, and serum cholesterol—all normal. A hateful word. Serum magnesium — not even mentioned. My lab must be slipping. I was getting nowhere fast. Where were the blood counts, smears, and sed. rates? I reached for the buzzer again.

Marilyn shimmied in. I could see her bosom heaving with each heart beat. But I was not auscultating. I had a job to do, and I had to do it fast. "Blood morphology," I commanded, "and throw in the sternal marrow." Breaking the sound barrier she threw the reports on my desk. Normal and borderline again. Not a hint.

Before I had time to summon her, as if reading my mind, Marilyn slunk in with more reports. Then she slapped my face. That girl could read my mind all right. But I wasn't being side-tracked. Quickly I glanced through the urinalysis, the C.S.F. report and the B.M.R. Nothing there. Vital capacity and circulation time — within normal limits, E.E.G. — alpha rhythm. I was stumped. I was stymied. Where do I go from here? There was nothing left for me to do, unless? Yes, I knew it had to come. The thing I dreaded — a physical examination. I rang again.

Marilyn tiptoed in, her crimson lips pouting. But I was in no mood for osculation. "Bring in the patient," I whispered hoarsely, "stripped for examination." Sensing my tension she darted out and returned with the patient in a wheel chair. I could hardly believe my eyes. "Not you," I cried, "it's the patient who is to be stripped." Quickly she unveiled him. Reluctantly I proceeded to examine. What a chore! The patient was not co-operating. He cried when my fingernails dug into his right upper quadrant. He kicked when the sharp blade of my pocketknife stroked his sole for a Babinski. He swore when the reflex hammer hit him hard on his patella. He screamed when my ear speculum touched his eardrums. He jumped sky-high when I tested his testicular sensation with my forceps. He was hostile throughout. But I was past caring. I had a diagnosis to make and it eluded me. The stethoscope was bringing in unpleasant noises. Must be the rustling of the interposed shirt. I was defeated. I was deflated.

Then it happened! Marilyn undulated in noiselessly. My head was swimming. But I was keeping it above water. She sensed the drama within me. "How about a history, Doc?" she whispered caressingly into my ear. This I never thought of. Repugnant as it was, I knew it was my last hope. Harsh realities call for harsh measures. I braced myself for the ordeal. With jaws set, through clenched teeth, I spat out: "What is your trouble?" The answer was a bombshell. "This is not what I came here to find out," he said without batting an eyelash. "What, then, did you come here for?" I cried utterly exasperated. "To get a premarital blood test," he said; "I'm getting married next week." Stunned by his reply I glanced quickly through the reports. Everything was done. Everything but a blood Wasserman. I broke out in a cold sweat. My hand shook as I pressed the buzzer again.

Marilyn strode in, vibrant and exciting, a burning volcano. I extinguished it fast. "Where's the Wasserman report?" I thundered. She paled, she trembled, she mumbled excuses. But she knew and I knew that it was all over between us. What happened was bigger than both of us. No one can omit number 38 routine lab test and stay on. She put on her coat and gloves. Tears were streaming down her cheeks. "So long, Doc," she sobbed, "it was fun while it lasted." I blew my nose. I bit my lip. I said nothing. Something big and important had gone out of my life. . .

I looked at my watch. My heart sank. The effort was wasted. It was late. Much too late to make the Dinner Meeting of the Hippocratic Society.

...the proof of the Patrician "200" is in the radiograph!

When you choose x-ray for private practice, look at performance as well as the price tag. "Economy" that is gained by short-cuts in table design or a reduction in power may mean slow exposures, blurred radiographs and repeated retakes. General Electric's Patrician "200" combination is designed with adequate power for private practice — a full 200 ma to stop anatomical movement sharply and clearly. Many other features found in larger installations are engineered into the Patrician: 81" table, independent tubestand, shutter limiting and automatic tube protection, to name just a few. And, considering its *uncompromising* G-E quality, this Patrician "package" is remarkably low priced.

Rent the Patrician through the G-E Maxiservice® plan that provides the complete installation, *including* maintenance, parts, tubes, insurance, local taxes — *everything* in one monthly fee. Get details from your G-E x-ray representative listed below.

Progress Is Our Most Important Product

GENERAL ELECTRIC

DIRECT FACTORY BRANCHES

EDMONTON, ALBERTA
10343 123rd St. • HUnter 8-9278

WINNIPEG
565 Portage Ave. • SPruce 5-2555

RESIDENT REPRESENTATIVES

CALGARY, ALBERTA
W. W. WHITE, 61 Walnut Dr. • CHurchill 9-4336

REGINA, SASKATCHEWAN
E. R. BAXTER, 1233 Jubilee Ave. • LAkeside 2-7314

SASKATOON, SASKATCHEWAN
R. A. MURIE, 2333 Hanover Ave. • DIckens 3-2006

Book Review

"Infectious Diseases of Children." Krugman-Ward, 2nd Edition, 1960. C. V. Mosby Co. Price $13.00.

The fact that a second edition of this book became necessary within two years should be evidence enough that this book has found a secure place for itself in the libraries not only of paediatricians but of medical practitioners everywhere. It is, by all odds, the most practical and helpful book available on the subject of infectious diseases in children. Advances in the field of immunology and control of infectious and communicable disease in infancy and childhood have been phenomenal within the last few years. One needs to mention only the availability of Salk vaccine for poliomyelitis, the use of hyperimmune vaccinal gamma globulin, hyperimmune pertussis serum, the development of oral attenuated poliomyelitis vaccine, the development of a new, effective measles vaccine, and the rapidly expanding knowledge of virus infections peculiar to childhood make this one of the most exciting areas in paediatrics. This book is beautifully printed and well illustrated with some excellent graphic charts illustrating the classical picture of the various infectious diseases. Special attention has been paid to the problem of rabies which has become a most important problem all over the country, and specific instructions are given in regard to the management of children who are bitten by potentially rabid animals. The very first chapter in the book deals with adeno-viral infections and contains an excellent bibliography. There is much new material on the management of acute bacterial meningitis where again the development of new treatment plans have resulted in a tremendous reduction in mortality within the last ten years. No one who has any clinical contact with infants and children should be without this book. It is highly recommended. — H. M.

Local Doctor Honoured

On Monday evening, November 7th, Deloraine and district gathered at a dinner meeting in the United Church to pay tribute to one of Deloraine's best known and most popular citizens, Dr. J. H. Buchanan.

The dinner was sponsored by the Deloraine Chamber of Commerce, the W.I., the Deloraine Agricultural Society, and the Deloraine Curling Club. One hundred and sixty-five guests were present, but lack of table space prevented many more from attending.

M. S. Colquhoun, chairman for the evening, introduced the guest speaker, Judge J. M. George of Morden, who, as a former member of the hospital board, was associated with Dr. Buchanan for many years.

Dr. J. H. Buchanan was born March 1st, 1881, in Huron County, Ont. He received his high school and model school education at Seaforth and Goderich. Teaching school for almost five years in Ontario, he came west and took normal training in Winnipeg in 1906, and taught school in Manitoba for three years. In 1909 he entered Manitoba Medical College, graduating in 1914 with the degree of physician and surgeon.

He spent a year and a half as senior surgeon, and a year doing medical work in the Winnipeg General Hospital. Later, he practiced at Steinbach, Holland and Miami. In 1948 he received life membership in the College of Physicians.

Dr. Buchanan came to Deloraine in 1918. During his years of practice, he has lost count of how many babies he has delivered, but the number is well over two thousand. The first birth he attended at Deloraine was that of Joe LePoudre, who was present at the dinner. The last baby he delivered was the son of Mr. and Mrs. Earl Hartel, who were also present.

During Dr. Buchanan's early years of practice, there were no modern hospitals to help care for the sick people of the community. All the babies were born in the homes. Other illnesses also had to be taken care of in the home. This meant doctors had to drive miles over rough country roads, using horses during the winter. That meant driving day and night during any epidemic which might break out, without modern medicine to prevent the spread of contagious disease.

In 1922, the residence of the late George Paterson was bought and remodelled into an eight bed hospital. This lessened the long drives for Dr. Buchanan, except in cases of emergency.

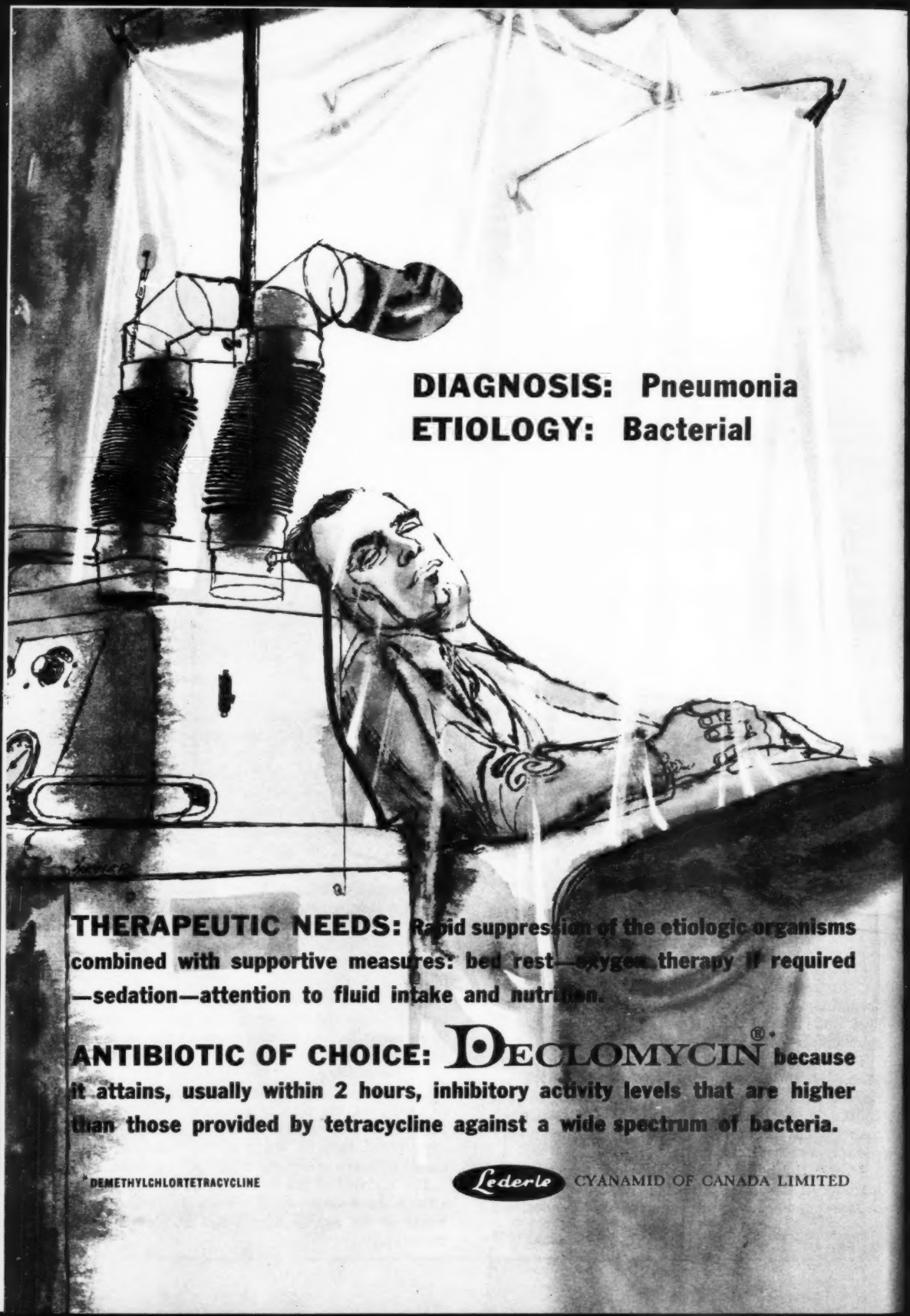
Dr. Buchanan's wife, Dorothy, and daughter, Lorna, now Mrs. Wm. Jardine, share his interest in medicine. Both are registered nurses.

Down through the years Dr. Buchanan has been noted for his interest in all sports, and especially in raising and racing fine harness horses, an interest he still maintains. This year a three year old colt he raised, now owned by Henry Maynard, paced her way to victory on major tracks in Manitoba. Among her wins was the award for the fastest mile raced this year at Deloraine, and the first heat of the Colt Futurity at Brandon.

Following the dinner, Dr. Buchanan was presented with a gold plaque and scroll containing the names of many of his former patients. His speech of acceptance was pleasantly spiced with the sense of humor and flair for story telling that has always made welcome company wherever he went.

The number of guests attending from a distance affirms the wide circle of friends Dr. Buchanan has made in his nearly fifty years of service to the community.

Reprinted from The Deloraine Times and Star, November 10, 1960.


DIAGNOSIS: Pneumonia
ETIOLOGY: Bacterial
THERAPEUTIC NEEDS: Rapid suppression of the etiologic organisms combined with supportive measures: bed rest—oxygen therapy if required —sedation—attention to fluid intake and nutrition.
ANTIBIOTIC OF CHOICE: DECLOMYCIN® because it attains, usually within 2 hours, inhibitory activity levels that are higher than those provided by tetracycline against a wide spectrum of bacteria.
*DEMETHYLCHLORTETRACYCLINE
Lederle
CYANAMID OF CANADA LIMITED

Association Page

Newsettes

M.M.A. Membership Fees

You can make the Treasurer a very happy man by submitting your Association fees now. A contribution to the Benevolent Fund is always in order.

The Treasurer's letter of January 3rd outlined the fee categories. If in doubt, call the Association office.

☆

Workmen's Compensation Board Schedule

This is a further reminder to all members that effective during the period January 1st, 1961 to May 15th, 1961, the Workmen's Compensation Board has agreed to a 40% increase in the fee schedule.

Negotiations are continuing with the W.C.B. in connection with a new schedule subsequent to the 15th of May, 1961.

☆

Annual Meeting — M.M.A.

It has been decided that scientific exhibits will be discontinued for the 1961 Annual Meeting.

☆

Proposed Regulations Under The Hospitals Act Relating to Standards for Licensure

The Manitoba Medical Association has been asked to make a critical survey of these proposed regulations. They have been drafted by the M.H.S.P. Standards Division and are contained in a 58-page manual.

In order that a report to the Executive may be available as soon as possible, an Ad Hoc Committee was set up under the Chairmanship of Dr. Dwight Parkinson. The committee is to be representative of the profession, as the proposed standards appear to have a bearing upon all aspects of medical participation in hospitals.

☆

Special Committee on Nursing

At the December meeting of Officers, this Special Committee was made a Standing Committee of M.M.A. and Dr. N. Merkeley was named Chairman.

☆

Extension of Out-patient Coverage under M.H.S.P

In November the M. H. S. P. circularized all practising physicians in Manitoba advising of the extension of out-patient coverage which became effective November 1st, 1960.

Some of the changes are extensions in coverage, others are new additions to the list which has been prepared and amended from time to time with the assistance of the Medical Advisory Committee.

There are now 47 items qualifying as insured services. An up-to-date list may be obtained from the M.H.S.P.

Speakers for District Society Meetings

This is a further reminder to District Societies that the Manitoba Medical Service will provide guest speakers on a Board or Management level to further acquaint members with M.M.S. affairs.

District Societies are asked to request speakers through the M.M.A. Office when arranging their scientific program.

☆

Medicare

The Minister of Health recently brought to the attention of Greater Winnipeg practitioners, some problems relating to the provision of drugs for Medicare patients. The Minister has requested that each prescription should indicate if a Medicare case, recording the group and contract number from the Medicare card.

A reminder was also given that in the Greater Winnipeg area the filling of a prescription by a local pharmacist is reserved for emergency or special circumstances.

☆

Accident and Sickness Claim Forms

This is a reminder that the combined forms (CMHIA-1), approved by the C.M.A. and the Canadian Health Insurance Association, are obtainable from the M.M.A. office upon request.

We have received inquiries about Assignment forms. These are not provided through C.M.A. and it is suggested that the wording be used as contained in the "Procedure Booklet for the Use of Accident and Sickness Claim Forms."

☆

Questionnaires, Surveys, Inquiries, Etc.

It has been brought to the attention of this office that members occasionally receive extensive questionnaires, surveys or inquiries from organizations requesting information.

It is suggested that members do not complete these circulars unless there is indication of endorsation by the Canadian Medical Association or the Manitoba Division. If in doubt, a call or letter to the M.M.A. office would verify whether or not the circular had been approved.

☆

C.M.A. Convention — 1962

More convention news — this time of national interest. In view of the fact that Manitoba will be the host for the 1962 C.M.A. convention the C.M.A. president-elect will be named by this Division during the course of this year.

Royal Commission on Health Services

You have probably seen releases in the newspapers concerning the Prime Minister's appointment of a Royal Commission to investigate and report on health services in Canada.

This action was based on a direct request from the C.M.A. for such a Commission following a strong recommendation from the C.M.A. Economics Committee.

The five-man Royal Commission will be headed by Chief Justice Emmett M. Hall of Saskatchewan's Court of Queen's Bench.

☆

Doctor Vergil Slee

Dr. Vergil Slee of the Commission on Hospital and Professional Activities, Ann Arbor, Michigan, was a luncheon guest of the Officers on December 8th.

Dr. Slee outlined the work of the Commission which was organized in 1950, and at that time supported by the Kellogg Foundation. At the present time, the Commission is supported by the American College of Physicians, American College of Surgeons, and the American Medical Association. It is a non-profit scientific and educational group, with trustees from the American College of Physicians, the American College of Surgeons, American Hospital Association and others from independent hospitals.

Hospitals in many states are members of the Commission and they represent in hospital discharges, about one and a half million patients a year. These hospitals send in clinical record abstracts and the information is punched on I.B.M. cards which are sorted and tabulated to obtain the required statistical data. The Commission originally provided hospitals and medical staffs with statistical reports but further development now allows them to give these bodies comparison figures with other hospitals of similar size, etc.

Dr. Slee demonstrated the Professional Activities Study Case Abstract which he said could be completed in five minutes by a clerk in the medical records department of the hospital. The abstract is completed upon discharge of the patient. The chart is then sent to the Commission for compilation at a current charge of 25c each. The clerk in the medical records department does not supply information calling for medical judgment, but records factual information contained in the patient's chart.

Dr. Slee was visiting Winnipeg under the auspices of the M.H.S.P. to advise how he assesses work in hospitals which in reality is an extension of Tissue Committee work. Through the Commission's program, Dr. Slee pointed out that it was possible to evaluate the quality of medical care being given in hospitals. Dr. Slee felt that with the information and equipment that M.H.S.P. now had, that a similar system could be established in Manitoba. Dr. Slee pointed out that the Commission only enters into contracts with hospitals, when presented with evidence that the medical staff is favorable to participation.

At the present time there are no Canadian hospitals participating in Dr. Slee's program.

☆

Important Notice—Group Life Insurance

You were advised last month that your executive had made arrangements for a revised formula in connection with the Group Life coverage afforded to the members through the Travelers Insurance Company.

You will receive from the Manitoba Medical Services a brochure, explaining the new formula in detail along with an application card. It is very important that this card be signed and returned at once, by those members who wish to take advantage of the new formula. We hope to have returns from a sufficient number of members to put the additional coverage in effect by March 1st, 1961.

Additional information may be obtained by contacting C. C. Murphy of the Travelers Insurance Company, WH 3-8475.

Note: There was a typographical error in the article on Group Life Insurance in the January issue—the third line should read "to increase the present life insurance coverage **of** $15,000.00 — not **to** $15,000.00.

R.P.H.S.

☆

Visiting Speakers

Department of Pediatrics, Faculty of Medicine University of Manitoba

Dr. George Wheatley, New York City, President of the American Academy of Pediatrics and Vice-President of the Metropolitan Life Insurance Company, will be a visitor with the Department of Pediatrics, Winnipeg Children's Hospital, on February 28th and March 1st, 1961. Dr. Wheatley will hold an afternoon conference on School Health and will speak to the residents and staff on problems of accident prevention. He is also scheduled to address the Annual Meeting of the Board of the Children's Hospital on "The Future of Children's Hospitals in the Country."

Dr. Gordon Millichap, Pediatric Neurologist, Mayo Clinic, Rochester, will be visiting the Department of Pediatrics on March 15th and 16th, 1961. Dr. Millichap is widely known for his work in the diagnosis and treatment of convulsive disorders in children and has done important experimental work in connection with febrile convulsions. Dr. Millichap will attend conferences and present lectures on various aspects of Pediatric Neurology during his stay.

Members of the Profession are welcome to attend any of the Rounds addressed by these speakers.

You are invited to our . . .

Annual Valentine Party

Reception - Dinner - Dance

Saturday, February the 11th, 1961

ROYAL ALEXANDRA HOTEL

6.00 p.m. - - Dress Optional

Join us. Meet your friends and enjoy every minute of a most sociable evening.

Make your reservations early. Further information may be obtained from the Social Committee.

Sponsored by: The General Practitioners' Association of Manitoba

College of Physicians and Surgeons of Manitoba

The Auditor's Report

Report and Accounts, September 30, 1960

To the Members of Council,
The College of Physicians and Surgeons of Manitoba,
Winnipeg, Manitoba.

Dear Sirs:

We have examined the accounting records of the College of Physicians and Surgeons of Manitoba for the year ended September 30, 1960, and have obtained all the information and explanations we have required:

Exhibits

"A" Statement of Funds as at September 30, 1960.
"B" Statement of Receipts and Disbursements for the year ended September 30, 1960.
"C" Particulars of Receipts — Current Account — for the year ended September 30, 1960 — with comparative figures for 1959.
"D" Particulars of Disbursements — Current Account — for the year ended September 30, 1960 — with comparative figures for 1959.

We submit the following comments relative to our examination and verification of the assets set forth in the Statement of Funds — Exhibit "A".

Investments

We attended at the safety deposit vault of The Toronto-Dominion Bank on October 4, 1960, together with Dr. M. T. Macfarland and Dr. T. H. Williams, and examined Province of Manitoba, Hydro Electric Power Commission of Ontario, and Canadian National Railways bonds of a par value of $70,000.00, as shown under the heading of Investment Account; and Province of Manitoba, Hydro Electric Power Commission of Ontario and Government of Canada bonds of a par value of $29,100.00 as shown under the heading of Gordon Bell Memorial Fund.

Changes during the year in the investments held are as follows:

Investment Account

Purchased:
Canadian National Railways bonds,
$4,000.00 5½% December 15, 1964 ..... $3,940.00

Gordon Bell Memorial Fund

Purchased:
Province of Manitoba bonds,
$1,000.00 5½% June 1, 1969 ..... 1,020.00

Bequests from Estate of Dr. Hugh MacKay

Government of Canada bonds,
$2,100.00 4¼% September 1, 1972 ..... $2,100.00
Hydro Electric Power Commission of Ontario bonds,
$2,000.00 4% March 1, 1966 ..... $2,000.00

All interest has been duly accounted for on the books of the College on a received basis.

Funds on Deposit

We received a certificate from The Toronto-Dominion Bank confirming the amounts on deposit at September 30, 1960.

Receipts and Disbursements

Disbursement cheques were signed by two authorized signatories, namely, Dr. M. T. Macfarland and Dr. T. H. Williams. Vouchers for expenditure bore the approvals of Dr. M. T. Macfarland and Dr. A. R. Birt or Dr. R. E. Beamish or Dr. A. L. Paine. Grants, donations, changes in salaries and certain other expenses were found to be in accordance with authorizations in the minutes of Council and Executive Committee.

In our opinion, the accompanying Statement of Funds and Statement of Receipts and Disbursements are properly drawn up so as to exhibit a true and correct view of the state of the affairs of the College as at September 30, 1960, and the result of its operations for the year ended on that date, according to the best of our information and explanations given to us and as shown by the books of the College.

In conclusion, we wish to express our appreciation of the facilities afforded and the co-operation extended to us during the course of our examination.

Yours very truly,
S. M. MILNE & CO.,
Chartered Accountants.

FINANCIAL STATEMENTS

Statement of Funds as at September 30, 1960

EXHIBIT "A"

	Investment Account	Current Account	Total	Gordon Bell Memorial Fund
INVESTMENTS				
Bonds — carried at par value:				
Province of Manitoba 4¼% due Oct. 1, 1968	$ 6,000.00		$ 6,000.00	
5½% due June 1, 1959				$ 1,000.00
The Hydro Electric Power Commission of Ontario:				
4% due July 15, 1974	40,000.00		40,000.00	
5% due Nov. 15, 1974/76	15,000.00		15,000.00	24,000.00
4% due March 1, 1966				2,000.00
Canadian National Railways: 5% due May 15, 1968	5,000.00		5,000.00	
5½% due Dec. 15, 1964	4,000.00		4,000.00	
Government of Canada: 4¼% due Sept. 1, 1972				2,100.00
	$70,000.00		$70,000.00	$29,100.00
UNINVESTED FUNDS				
On deposit with The Toronto-Dominion Bank	2,158.52	4,551.05	6,709.57	664.30
Petty cash fund		10.00	10.00	
	$72,158.52	$ 4,561.05	$76,719.57	$29,764.30
DEDUCT— Deposits on applications pending		(115.00)	(115.00)	
SURPLUS	$72,158.52	$ 4,446.05	$76,604.57	$29,764.30
Approximate quoted market value of above bonds as at Sept. 30, 1960	$65,802.50			$28,562.00

Note: With the exception of the bonds received from the Estate of Dr. Hugh MacKay, namely $2,000.00 Hydro Electric Power Commission of Ontario 4% March 1, 1966, and $2,100.00 Government of Canada 4¼% September 1, 1972, which are bearer bonds, all securities are registered in the name of The College of Physicians and Surgeons of Manitoba.

STATEMENT OF RECEIPTS AND DISBURSEMENTS
For the Year Ended September 30, 1960

EXHIBIT "B"

	Investment Account	Current Account	Total	Gordon Bell Memorial Fund
CASH RECEIPTS				
Interest on bonds	$ 2,965.00		$ 2,965.00	$ 1,362.62
Interest on bank accounts	40.56		40.56	13.35
Fees and other receipts — per Exhibit "C"				
Donations		25,160.23	25,160.23	
F. W. Duval, M.D., F.R.C.S. (C)				325.00
Estate of Dr. Hugh Mackay (Bonds having par value of $4,100.00 not included)				214.52
	$ 3,005.56	$25,160.23	$28,165.79	$ 1,915.49
CASH DISBURSEMENTS				
General disbursements per Exhibit "D"		$26,944.32	$26,944.32	
Scholarship award Dr. F. A. Herbert				$ 800.00
Grant to Medical Library Committee	$ 750.00		750.00	
Grant to Manitoba Medical Association for extra-mural post-graduate work	500.00		500.00	
Manitoba Medical Association, expense of Workmen's Compensation Board fee taxing committee	30.00		30.00	
Purchase of bonds— $4,000.00 Canadian National Railways 5½% Dec. 15, 1964	3,940.00		3,940.00	
$1,000.00 Province of Manitoba 5½% June 1, 1969				1,020.00
	$ 5,220.00	$26,944.32	$32,164.32	$ 1,820.00
Net receipts or (disbursements) for the year	(2,214.44)	(1,784.09)	(3,998.53)	95.49
Add: Cash in banks and on hand at commencement of year	4,372.96	6,345.14	10,718.10	568.81
Cash in banks and on hand as at Sept. 30, 1960 per Exhibit "A"	$ 2,158.52	$ 4,561.05	$ 6,719.57	$ 664.30

PARTICULARS OF RECEIPTS — CURRENT ACCOUNT
For the year ended September 30, 1960

EXHIBIT "C"

(with comparative figures for 1959)

	1960	1959
Registration fees	$ 8,690.00	$10,140.00
Annual fees	11,336.89	10,904.00
Certificates:		
M.C.C.	938.39	1,250.00
G.M.C.	35.12	65.00
Temporary licenses	560.00	445.00
Interne enrollment	566.85	185.00
Specialist registration fees	450.00	215.00
Documentation fees	1,925.03	2,482.28
Medical students registration fees	39.00	34.00
Sales of register and mailing list	502.00	469.00
Credentials committee — University of Manitoba	60.00	90.00
Miscellaneous income	1.95	.24
Deposits on applications pending	55.00	88.41
	$25,160.23	$26,367.93

PARTICULARS OF DISBURSEMENTS CURRENT ACCOUNT
For the year ended September 30, 1960

EXHIBIT "D"

(with comparative figures for 1959)

	1960	1959
Salaries	$15,854.32	$10,743.69
Meetings:		
Annual	738.50	856.90
Special	752.50	781.90
Executive committee	295.20	367.60
Special committees	1,242.07	808.40
Luncheons, annual and special meetings	217.75	170.25
Janitor service, annual and special meetings	10.00	10.00
Documentation fees paid to University of Manitoba	75.00	105.00
Manitoba Medical Association for office rental, secretarial services, etc.	2,040.00	2,025.00
Stationery and office supplies	356.37	282.71
Printing — Medical Register (1,500)	1,929.18	
Printing	692.40	724.19
Postage	472.52	448.15
Insurance Premium	56.00	56.00
Audit fee	175.00	175.00
Legal Fees	790.59	370.00
Miscellaneous, office and general expenses	900.17	287.49
Bank exchange	64.25	29.23
Expenses of registrar — out of town meetings	233.00	225.00
Deposits refunded on unaccepted applications	40.00	439.00
Manitoba Medical Association, expense of Workmen's Compensation Board fee taxing committee		120.00
Office equipment	109.50	
Grant to Manitoba Medical Association for extra-mural post-graduate work		500.00
	$26,944.32	$19,525.51

Note: The grant to Manitoba Medical Association of $500.00 for extra-mural post-graduate work and the assessment for Workmen's Compensation Board fee taxing committee of $30.00 for the year ended September 30, 1960, was paid from Investment Account as shown on Exhibit "B".

☆

Announcement

Schering Corporation Ltd. announces that effective January 16, 1961, the distribution of the products of White Laboratories of Canada Ltd. will be consolidated with that of the Schering line.

In line with Schering's expansion program, this move is intended to better serve the medical and pharmaceutical professions by extending from coast to coast the distribution of recently acquired "WHITE" products.

New additions to the Schering product line will provide the medical profession with specialties of the highest standard for which a wide range of applications is encountered in everyday practice.

New additions to the Schering line will be distributed through established Schering wholesalers.

Social News

Reported by K. Borthwick-Leslie, M.D.

Roses — Cornish hens (under glass??)—nope—Better! — Wild Rice — and you know the price of that. Wine—Floor show—Irvin Plumm's Orchestra. Short but short speeches. Where? When? General Practitioners' Association Annual Valentine Party, Royal Alexandra Hotel, Saturday, Feb. 11th. Come one, come all for a wonderful evening. Hope to see you.

☆

For this I am completely at a loss for words. I'm so proud of Patricia Blondal and her novel: "A Candle to Light the Sun" and so completely in accordance with Derek Crawley's review, who states simply, "Canada has lost, not a promising novelist, but an obvious genius." Personally I've not had enough nerve yet to read Pat's book—her loss as yet hurts too much.

It seems an amazing coincidence that the same week Pat's book appeared in Winnipeg, her husband, Dr. Harold Blondal and his co-workers from Montreal were reporting to the Royal College of Physicians and Surgeons in Ottawa the results of their work with N-Dichloracetyl-DL-Serine — an anti-cancer drug developed by them in the Charles E. Frosst Laboratories.

Harold's T.V. interview was very impressive and sincere. Congratulations and good wishes for further success in this dedicated work.

☆

Dr. Marie Storrie is at present attending the Ontario Heart Foundation, Toronto, annual meeting. She is presenting a paper, reporting the investigation of two hundred cases she and Dr. Robt. Beamish have just finished. Topic — Impending Myocardial Infarction.

☆

The Canadian Arthritic and Rheumatic Society has announced the awarding of a $3,000 scholarship to Dr. Wm. Chodirker of Winnipeg General Hospital, for further study in Burlington, Vermont.

☆

Dr. Alvin Zipursky, U. of M., has been appointed medical director for the Manitoba Red Cross Transfusion Service.

☆

Dr. and Mrs. Glen Lillington, of Palo Alto, Calif., stopped in Winnipeg in transit to Ottawa, where Dr. Lillington delivered a paper to the Royal College of Physicians and Surgeons of Canada.

Dr. and Mrs. Sam A. Boyd announce the engagement of their only daughter, Lynne Maureen to Mr. Miles Pepper of Montreal. The wedding will be in Knox United Church, Saturday, February 11th.

☆

Dr. and Mrs. M. T. Macfarland announce the engagement of their only daughter, Frances Murray, to Dr. Alastair Harris Imrie, son of Dr. and Mrs. D. S. M. Imrie of Edinburgh, Scotland.

☆

The engagement is announced of Miss Josephine Hyde to Mr. Earl R. Stephenson, only son of Dr. and Mrs. Earl Stephenson. The wedding will be March 4th in the Crescent Fort Rouge United Church.

☆

Born on January 8th at Castlegar, B.C., to Dr. Waldo M. and Mrs. Yule—Joanne Elizabeth, sister for Geoffrey.

☆

Dr. and Mrs. Russel Popoff are pleased to announce the birth of their son, Brian David, on New Year's Day, 1961.

☆

Dr. and Mrs. G. F. Kremer announce the arrival of Jeffrey Frederick, Jan. 8, 1961.

☆

Dr. and Mrs. J. E. Bennett also welcomed their son, Timothy John, Jan. 15, 1961.

☆

Dr. and Mrs. John Sawchuk, Rolette, N.D., announce the birth of a son — Theodore John — Jan. 12, 1961.

☆

Dr. and Mrs. Max Cham are happy to announce the arrival of Susan Miriam, January 24th—baby sister for Bonnie and David.

☆

Dr. and Mrs. J. G. Wade also welcome their daughter, Marianne Elizabeth, January 23, 1961.

☆

Did you hear about one of our members occasionally afflicted with insomnia? She was persuaded by one of our Hypnosis enthusiasts to try his instructions, and very successfully too. That night she composed herself, relaxing but concentrating on that uplifted hand. Sure enough, slowly, sleep engulfed her — but whom — the fist came down kerplunk on her face! Awake for the night. C'est vie.

HOSPITAL MEETINGS

Hospital	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
Children's	12:00 Surgical Rounds	10:00 Staff Rounds "B" Service 12:00 Postgraduate Seminars	9:30 Staff Rounds "C" Service 11:00 Clinical Path. Conference	11:00 Grand Ward Rounds	10:00 Staff Rounds "A" Service	9:00 Newborn Conference
Deer Lodge	Clinical Luncheon (1st Monday)			11:00 Ward Rounds		
Grace	12:00-2:00 p.m. Weekly Seminar	Clinical Luncheon (3rd Tuesday)		12:00-2:00 p.m. Weekly Seminar		
Misericordia		2. Clinical Luncheon				
Municipal			7:30 p.m. Review of Deaths (2nd Wednesday)			8:30 Clinical Staff Conf. and Ward Rounds
St. Boniface	11:00 Paediatric Rounds	11:00 Surgical Rounds	11:00 Grand Rounds 11:00 Cardio-Pulmonary Conf. (2nd Wednesday)	8:00 Orthopedic Rounds 11:00 Tumor Clinic 11:00 Obstetrical Rounds 12.u0 Clinical Luncheon (2nd & 4th Thurs.	11:00 Medical Rounds 11:00 Cardio-Pulmonary Conf. (4th Friday)	
St. Boniface Sanatorium		12:30 Clinical Luncheon (2nd Tuesday)				
Victoria		Tissue Committee (1st Tuesday)			3. Clinical Luncheon 4. Active Med. Staff	
Winnipeg General		11-12:00 Medical Ward Rounds	9-10:00 Tumor Clinic 11:30-12:30 Chest Conference	12:15 Clin. Lunch. (1st & 3rd Thurs.)	11:30-12:30 Neurological Conference 4:00-5:00 Surgical & Service Rounds	10:00 Surgical Conference 11:00 O.P.D. Conference 11:00 Obstetrics & Gynaecology Conference
Brandon General			Medical Staff Lunch (Wed. prior to 2nd Tues. each month)			10:00-noon Clinical Drs. from S.W. Manitoba invited (Nov. to May)

DEPARTMENT OF HEALTH & PUBLIC WELFARE

COMMUNICABLE DISEASE PICTURE

North of 53 District

No communicable diseases of any interest reported.

Northwestern District

Other than four cases of scarlet fever, no reports of interest were received.

Northern District

Twenty-five cases of whooping cough were reported. One case of scarlet fever and one case of paratyphoid.

Brandon District

Ten cases of infectious hepatitis; one meningococcal infection; 25 scarlet fever were reported.

Central District

One case of scarlet fever was reported.

Winnipeg District

Reports were received for one case of undulant fever; two cases of diphtheria. One was a girl from the Ste. Anne district, who had been immunized originally against the disease in 1953, and had received a booster in 1959. The second case was a nine year old boy from Hanover, who had been immunized in 1959 and given a booster in 1960. Reports were received also on eighteen cases of infectious hepatitis. One meningitis due to an ECHO virus; one meningitis due to Coxsackie virus and two mengitides due to Other and Unspecified causes. One case of poliomyelitis was reported in a seven month old girl, who had received no Salk vaccine. There were two cases of pertussis; two of scarlet fever and one case of typhoid fever was reported in a three year old male, from West Kildonan.

Southern District

Nil.

LIST OF DEATHS FROM COMMUNICABLE DISEASES
December, 1960

URBAN: Cancer, 103; Influenza, 3; Meningitis (meningococcal), 2; Pneumonia, Lobar (490), 5; Pneumonias (other forms), 36; Tuberculosis, 8; Bacillary dysentery, 1; Late effects of acute poliomyelitis, 1. Other deaths under 1 year, 34. Other deaths over 1 year, 424. Stillbirths, 46. Total, 663.

RURAL: Cancer, 32; Influenza, 2; Meningitis (meningococcal), 2; Pneumonia, Lobar (490), 3; Pneumonias (other forms), 14; Tuberculosis, 6; Bacillary dysentery, 1. Other deaths under 1 year, 17. Other deaths over 1 year, 186. Stillbirths, 22. Total, 285.

INDIANS: Meningitis (meningococcal), 1. Other deaths over 1 year, 5. Stillbirths, 1. Total, 7.

Unorganized Miscellaneous

One case of diphtheria was reported from Washow Bay, in a seventeen year old female, who had received no immunization since 1948.

General

The incidence of communicable diseases in general was quite low. Cases of diphtheria have been occurring sporadically and there have been fourteen such cases during 1960. The incidence of paralytic poliomyelitis has not been high during 1960 and thirteen cases were reported.

Effective against most bacteria even penicillin resistant strains. Speedy, safe relief from soreness. Pleasantly flavoured—no antiseptic 'after-taste'.

DEQUADIN LOZENGES

In tubes of 20 and dispensing pack of 250. Each lozenge contains 0·25 mg Dequadin (dequalinium) chloride in a flavoured sucrose base.

Dequadin has a wide antimicrobial spectrum and even at low concentrations inhibits the majority of skin pathogens. Effective against antibiotic-resistant staphylococci, it diminishes the risk of resistant strains developing.

GLAXO-ALLENBURYS (CANADA) LTD, 52 BARTOR ROAD, WESTON (TORONTO) ONTARIO

Library Corner

To Bind or Not to Bind

Periodicals are the backbone of a medical library. So much so, that the University of Manitoba Medical Library spends nearly ten times as much on subscriptions and binding as on books. At the present time we receive nearly five hundred titles, and of these about half are currently bound.

To some extent, a medical library is evaluated by the percentage of periodicals that are regularly bound. When a survey team, representing the Evaluation Council on Medical Education and Hospitals of the American Medical Association, and the Association of American Medical Colleges, inspected the Manitoba Medical Library in 1957, it made strong recommendations to increase the binding list, pointing out that, if a periodical is worth buying, it is worth preserving. Single issues are more easily lost or damaged than bound volumes, and therefore great emphasis must be placed on having them bound as quickly as possible.

The average borrower, if he thinks about it at all, probably imagines that periodicals go from the shelves to the bindery without much effort on anyone's part. This is a mistaken impression, for a good deal of time and processing is often necessary before a medical journal can be properly bound. To begin with, the volume must be complete, with title page, table of contents and indices. Many times when a title is withdrawn for binding, it is found that one or more issues are missing, or out on loan. Then it is the duty of the Circulation Department to try and locate the strayed number on the library shelves, or to call it in if it is overdue. Sometimes a borrower finds he has lost a periodical belonging to the Library. This may not seem a serious matter to him, as he thinks he can remedy the situation by offering to pay for a replacement. What he may not realize is that replacement depends on locating a duplicate copy, and this is often very difficult to do. The cost of publishing a medical journal is so high that many publishers make a practice of limiting their output to a subscription list. Frequently it is found that the wanted issue is out of print. It is then necessary to search catalogues, and to check the Medical Library Exchange lists, perhaps for months or years before a copy can be found, and often it is never located. While this search is in progress, that volume is either out of circulation, or it is left on the shelf unbound, where it runs the risk of being further depleted or damaged.

When the material to be bound is complete, further processing takes place. Each page must be examined for omissions, duplications, tears, marks and corrections. Modern inventions have greatly speeded up the work of repair. What used to be a lengthy and difficult job can now be done with ease and dispatch by employing plastic liquids and tapes. However, there is one modern invention that is not always appreciated by librarians, and that is the ballpoint pen. Before its advent, borrowers who felt the urge to doodle, underline, or write remarks on periodical pages, usually did so in pencil, which is comparatively easy to erase. Now the ubiquitous ballpoint is generally used, which makes the clean-up job much harder. Possibly the doodlings of famous men might, on occasion, enhance the value of a journal, but in general such practice tends to decrease the worth of the item so disfigured. It can also lead to unexpected consequences, such as the case of the page that had a severe criticism of one of its paragraphs jotted down in the margin, and below, in a different hand, was the single word "nuts!" One can imagine the ultimate appearance of the page if each succeeding reader decided to join the controversy by adding his own comment.

When repairs and corrections have been attended to, there are still further details to check before the volume can go to the bindery. The average length of time needed to process a volume for binding is one hour. Fortunately, this should lessen as time goes on and publishers become more aware of problems connected with binding. Twenty-five years ago many publishers ran plates to the edge of pages, printed title pages in the middle of issues with text on the other side, or omitted them altogether; sometimes there were no volume contents or indices, and pages or whole issues were so uneven in size that proper trimming would endanger the text. These and other binding difficulties have been pointed out to publishers from time to time, and in many cases they have been rectified.

Another problem in binding preparation is to determine the maximum desirable thickness for each book. If two or more volumes are bound together, it may mean that their use is limited, since one borrower may perforce remove from circulation two volumes when he only requires to consult one of them. In other instances, it may be considered necessary to split a volume because of its sheer physical bulk; for example, the British Medical Journal, which has fifty-two single issues in one year. The Medical Library attempts to bind the most widely used titles in single or double volumes, and to partially offset this expense by making thicker volumes of the less frequently consulted journals, wherever such practice is feasible.

There are a number of different grades of binding available, with considerable variation in price. The Medical Library requires the highest workmanship in order to ensure that its journals will be durably preserved and capable of withstanding maximum use. Each section must be first machine-stitched with linen thread, and then hand-stitched

in "gatherings." This holds the pages securely but allows the book to open flat. The binder must be meticulous in trimming to avoid mutilating text or plates. It is essential to use good quality linen reinforcing on the inside hinges, and only the finest gold leaf will produce lettering that will not fade. To prevent the spine from warping, the book must remain in the press for a considerable period of time, the longer the better — and also the more costly to the binder, since it holds up the use of this particular piece of machinery. Finally, it is vital that the library's instructions to the binder be followed completely and exactly, for each title and for each volume of each title that is bound. In order to obtain these standards, the Medical Library has found the most economical binding is the most expensive.

Airdrie Cameron,
Binding Assistant.

☆

Letter to The Editor

Dear Editor:

We, of the Swan River Valley medical group, would like this letter to represent an open letter to the Economics Committee of the Manitoba Medical Association, presenting the thoughts and opinions of a group of rural physicians regarding the "Medicare" program in this province.

We would like to state first that we are fully aware of our duties as medical practitioners to treat the needy and indigent regardless of ability to pay. We find, however, the "Medicare" plan has many shortcomings in a rural setting and we feel it favors city practitioners who unfortunately form the vast majority in this province.

A city physician sees and treats patients, covered by this plan, in their homes and in his office and receives remuneration, as we do. If his patients require hospital care or surgery they are admitted as public patients under the care of house physicians, which would do much to foster teaching programs. If a patient in the country, especially in an area as isolated as ours, requires hospital care under the "Medicare" plan, to avail himself of free medical care while in hospital, he must make a long and costly trip to a major centre, which usually means Winnipeg. On the other hand, he can be admitted to his own hospital, and his physician receives no remuneration for his care. We would point out again that we do not object to providing this care. However, we do feel that our city colleagues have an unfair advantage. We feel that the "Medicare" plan is of such little value to us that we would almost prefer to be without it since it only creates the feeling amongst "Medicare" patients that the plan should either be comprehensive, pay a fixed proportion of doctors' fees (as it does in Saskatchewan), or should not exist at all.

We would appreciate the Economics Committee keeping these points in mind during future negotiations with governments and other agencies. We also feel that rural practitioners should be represented in larger numbers on policy making committees.

We hope this letter provokes further discussion of this matter in other rural medical groups and societies.

Yours truly,
Drs. J. Honig, L. Jonat, B. Jonsson, Swan River;
R. F. Clark, Benito.

Detailmen's Directory

Representing Review Advertisers in this issue, whose names are not listed under a business address.

Ames Company of Canada, Ltd.
W. H. Davis ..... VE 2-4869

Arlington-Funk Laboratories, division U.S. Vitamin Corp. of Canada, Ltd.
Ed Lessor ..... HU 9-1841

Bristol Laboratories of Canada Ltd.
Boyd C. Affleck ..... CA 2-1154
Peter Bayley ..... VE 2-5984

British Drug Houses
W. S. Langdon ..... GL 3-1325
H. Harvey ..... TU 8-5341
D. W. (Don) Young ..... VE 2-0256

Canada Pharmacal Co. Ltd.
Doug Shaw ..... HU 9-7735

Ciba Company Ltd.
Leslie D. MacLean ..... CE 3-3240
Richard Loewen ..... CH 7-1017

Connaught Laboratories
Brathwaites Ltd. ..... WH 2-2635

Frosst, Charles E.
W. M. Lougheed ..... HU 9-3963
W. J. McGurran ..... AL 3-0722
E. R. Mitchell ..... HU 9-6164
R. P. Roberts ..... AL 3-4032

Glaxo-Allenburys (Canada) Ltd.
H. W. (Bert) Heaslip ..... TU 8-1807
Ed Tackaberry ..... HU 9-4184

Hoffman-La Roche Limited
Paul E. Prendergast ..... AL 3-1962
Oscar Lyseyko ..... GR 4-4402

Horner, Frank W. Limited
Jos. Lavitt ..... JU 9-1691
Wes. Reimer ..... ED 9-3851
James N. Best ..... GL 3-6820

Lederle Laboratories
W. C. Hall ..... HU 9-2207
Ted Smith ..... AL 3-0437
R. D. Pollard ..... SP 2-4723
Bob Duff ..... JU 6-3628

Poulenc Limited
J. B. Peters ..... VE 2-3862

Robins (Canada) Ltd., A. H.
Harold Tetlock ..... SP 5-5624
Fred Gallinger ..... ED 4-1307
Ted Sims ..... ED 9-3154

Sandoz Pharmaceuticals Ltd.
H. D. Robins ..... ED 8-8216

Schmid (Canada) Ltd., Julius
H. V. Walker ..... LE 3-8664

Searle & Co., G. D.
Harry Chambers ..... ED 4-0989
B. Bishop ..... ED 8-8824

Smith Kline & French Inter American Corp.
Aylmer A. Dunlop ..... CH 7-8534

Warner-Chilcott Labs.
A. L. (Andy) Argue ..... TU 8-1619
Gordon Harrower ..... AL 3-4147

Winthrop Laboratories
R. M. Kelly ..... Res. GR 5-6038
A. E. Pauwels ..... CE 3-2024

Wyeth & Brother, John
A. W. Cumming ..... HU 9-5694
Stuart Holmes ..... JU 9-4273